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OF

THE PROTESTANT EPISCOPAL CHURCH
IN PHILADELPHIA

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OF THE

EPISCOPAL HOSPITAL

VOLUME V

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The present volume, owing to the interruption of the war, is the first to be published since 1916.

The Committee of Publication thinks it proper to say that the Hospital holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in this volume.

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John B. Carson, M.D., *Chairman.*

This volume has been edited by
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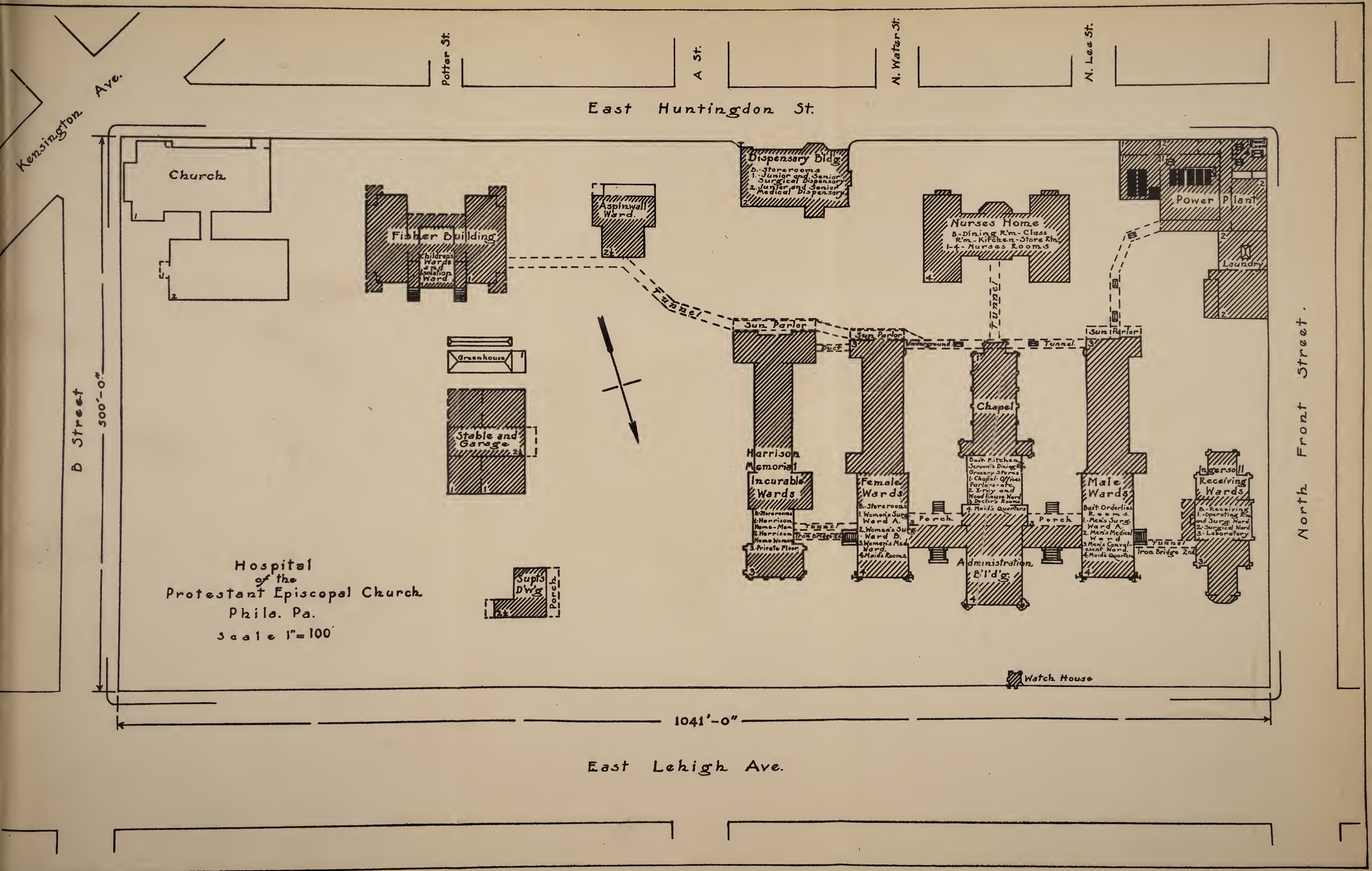

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Gill: The Relation of the Cripple to the Public.
Gill: A Case of Sprengel's Deformity—Congenital Elevation of the Scapula.
Gill: Surgery of Spastic Paralysis.
Gill: Deformities of the Hand.
Gill: Multiple Enchondromata of the Bones of the Right Hand of a Child Two and a Half Years of Age.
Gill: Dupuytren's Contracture, with a Description of a Method of Operation.

From the Pediatric Department of the Hospital

Sturtevant: Amyotonia Congenita.
Bender: A Case of Diabetes Insipidus in a Child Two Years of Age.

From the Syphilis Clinic of the Hospital

Walker: Inauguration of a Special Clinic for Syphilis.

From the Radiological Laboratory of the Hospital

Voegelin: Malpositions of the Colon, with Report of an Unusual Case.

From the Obstetrical Service of the Hospital

Parke: Cesarean Section in the Treatment of Eclampsia.
Parke: Operative Technic of Therapeutic Abortion.
Parke: Repairs Following Labor.

From the Staff of Base Hospital 34, A. E. F.

Ashhurst: Receiving and Operating Pavilions for a Modern Base Hospital.
Ashhurst: The Method of Recording Surgical Operations at the Front.
Ashhurst: Cases Illustrating Gunshot Wounds of the Vascular System.
Coleman: Report of the Dental Department.
Durham: The Treatment of Shock and Hemorrhage.
Jones: Experimental Implantation of Foreign Tissue Into the Lumen of Large Arteries.
Jones: Spontaneous Evacuation of a Gauze Sponge from the Peritoneal Cavity by Way of the Bowel.

FROM THE REPORTS OF THE SUPERINTENDENT FOR 1917–1919

	1917	1918	1919
Patients admitted	4,541	4,253	4,227
Patients remaining from previous year	343	299	307
Total number treated during year	4,884	4,552	4,534
Patients discharged	4,131	3,691	3,895
Patients died	454	554	352
Patients remaining December 31	299	307	287
Total	4,884	4,552	4,534
Av. daily number of patients in Hospital	329.75	314	294.33
Capacity of Hospital (beds)	500	500	500
Av. duration of treatment of in-patients excluding those in Harrison Memorial House for Incurables (days)	19	17	17.5
Cost of patient per day*	$2.07	$2.14	$2.92
Gross cost of patient per day (calculated on total expenditures of all kinds)	$2.40	$2.38	$3.25
New patients treated in the Dispensary:			
Medical	3,429	2,508	2,849
Surgical	6,403	4,740	4,550
New patients treated in the Dispensary:			
Medical	3,429	2,508	2,849
Surgical	6,403	4,740	4,550
Eye	2,815	2,162	2,153
Ear, Nose and Throat	1,862	1,236	1,572
Skin	480	341	450
Orthopedic	525	443	475
Genito-urinary	205	131	213
Dental	1,265	440	515
Pediatric	192	237	235
Syphilis	95	11	41
Receiving Ward	7,545	7,069	7,932
Total	24,816	19,318	20,585
Av. daily number of Dispensary patients	251	209.3	222.5
Av. daily number of Emergency Cases in the Receiving Ward	20.67	19.33	21.75

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* Cost of patient per day, as distinguished from gross cost, means the deduction of $12,000 for the Dispensaries and the deduction also of the Improvement and Repair Account. These figures include all patients admitted to the wards.

AMYOTONIA CONGENITA[1]

By CHARLES N. STURTEVANT, M.D.
PEDIATRIST TO THE HOSPITAL

As originally described by Oppenheim, amyotonia congenita consists of a flaccid paralysis, apparently congenital, most marked in the extremities, especially in the lower, and varying in a degree up to almost complete paralysis of the whole body. There is not necessarily any atrophy visible. Sensation is not disturbed, the tendon reflexes are diminished, the hypotonia is so great that there is usually passive mobility at the joints, and in bad cases the child is unable to sit and the head falls loosely in any direction.

Though but first discovered in 1900, there has since been much written about the condition, and many cases have been reported. A. Faber, in his review of the subject in April, 1917, was able to gather reports of 115 cases and 27 anatomic records; yet throughout the literature there appears an uncertainty regarding the etiology. Several possibilities are considered:

1. That there is some congenital failure of the development of the muscles, the cause being inherent in themselves.
2. That there is a failure of the peripheral motor neurons in their entirety to develop.
3. That there is a nutritional disturbance similar to rickets, which has to do with the metabolic processes of which no clear conception can yet be made.
4. That some derangement of the endocrin activities inhibit the growth of these muscles.

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[1] Read at a meeting of the Philadelphia Pediatric Society, April, 1918.

Each of these possibilities offers wide fields for research, but with none of them does the situation appear as hopeless as in the case of so many other congenital conditions. In fact, not a few of the cases of amyotonia congenita reported have improved markedly and grown to full development, though usually with some impairment of the muscular power. This is what adds zest to our hope of a solution of the problem and makes the study of amyotonia of particular interest.

The following case appeared in the Babies' Clinic at the Episcopal Hospital, Philadelphia, in the spring and summer of 1916, and was referred to the wards:

The patient was a female infant (F. F.), aged seventeen months, with the following history: The father and mother were each twenty-one years of age, living and in good health; American-born but of English ancestry. There was no family history on either side of tuberculosis, syphilis or anemia. The patient was an only child. The mother during her pregnancy was very nervous, crying a great deal and very much "run down." The fetal movements were weak; the labor quite severe, although not instrumental. Weight at birth was six pounds.

The mother was again pregnant when the baby first came under our observation, and has since been delivered, eighteen months after the patient's birth, of a perfectly normal baby. This second baby has been watched one year and shows no signs of any abnormalities similar to that of the first born. These points were observed with great interest, because clinical evidences of hereditary or reproductive defects would naturally be of much importance in the study of such a case. Faber found from a study of 60 cases that 53 per cent. were born of a fourth or later pregnancy, and suggested thereby the possibility of reproductive exhaustion as a factor in the production in this disease. The observation, however, that the fetal movements were weak coincide with his statistical study, and suggests, clinically, that the disease is probably present before birth.

From birth the child did not thrive and the mother noticed

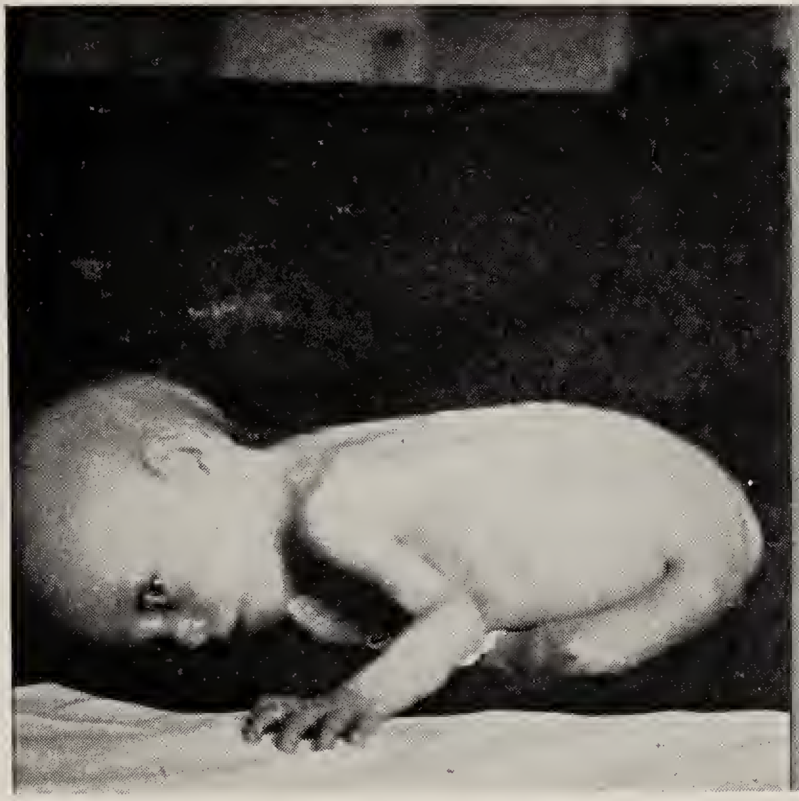

Fig. 1.—Case of amyotonia congenita, aged nineteen months.

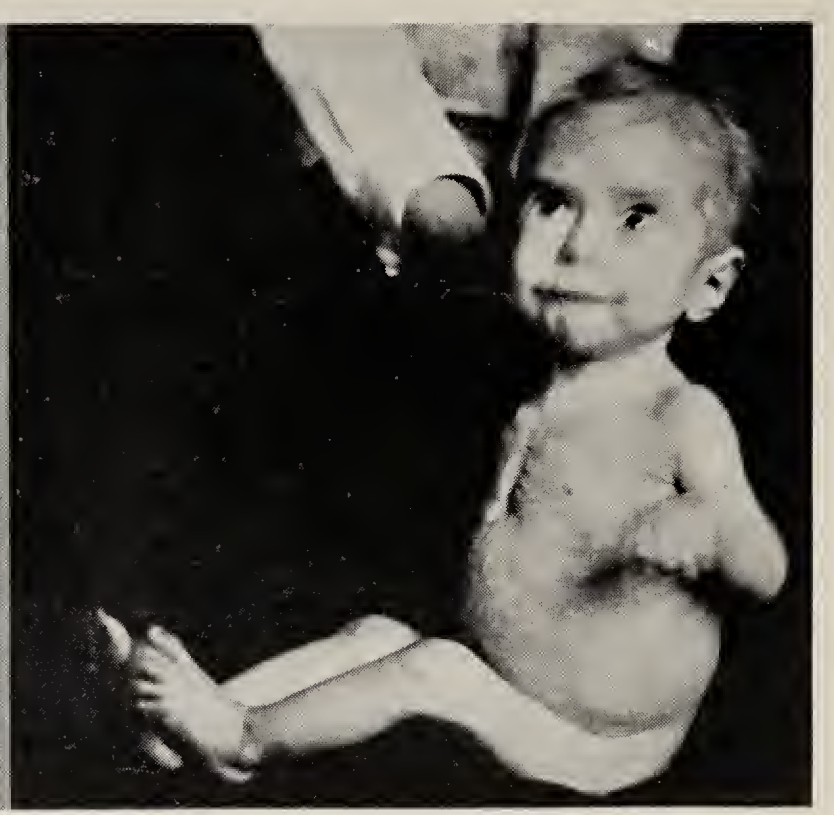

Fig. 2.—Case of malnutrition following enteritis, aged nine months. (Nurse is steadying only to prevent a lateral fall.)

from the onset extreme weakness of the muscles. In most of the cases reported in the literature the weakness has been present from birth. It frequently happens, however, that the parents do not notice the defect until it is realized that the baby is not sitting upright or standing or walking as other children of the same age are doing. Shoog reports one case not recognized until the fourteenth month, the lack of muscle power being thought but an exaggeration of the physiologic weakness of early infancy. A few cases have been reported as developing after illness as diarrhea, bronchopneumonia, measles, etc.

Our infant was breast-fed for two weeks, and then, because of insufficient milk, was weaned and given condensed milk for one month. This did not agree with the baby, and frequent attacks of vomiting and diarrhea induced the mother to change to a milk formula and later to a malted milk. The amount was regulated by the appetite of the infant, the interval by its crying. This improper feeding did undoubtedly increase the degree of muscular weakness, and was probably responsible for the emaciation our infant showed, but in no wise affects the diagnosis of amyotonia congenita. The classical description is that there is no visible muscular wasting because of the disposition of fat and a generous layer of adipose tissue. Several of the cases classified by Griffith and Spiller, however, showed muscular wasting.

The degree of hypotonia is pathognomonic. The accompanying photographs show the position assumed by the patient when an effort is made to make her sit upright and the position assumed by another infant in the ward. This second infant was ten months younger than the case here reported, but has, however, a similar degree of muscular wasting, due to a long-continued enteritis. It is to be noted that though there is muscular weakness in this second infant, there is some attempt to keep the spine erect successful to a degree not possible for a case of amyotonia.

The distribution of muscular weakness in our case conforms with the average distribution of the cases reported in the literature. The lower extremities are always involved. The upper extremities are usually involved, and next in frequency the trunk, as shown by the inability to sit upright or to hold the spine erect. The muscles of the abdomen are often involved, as shown by the abdomen being protuberant. It is frequently stated that the muscles of expression are not involved in amyotonia. This may be, however, because there is but a partial involvement, and there is still sufficient power to meet the not very exacting demands of facial expression. Ashly, in 1910, reported a case, aged eight years, occurring in the Manchester Children's Hospital, England, showing marked involvement of the face, which permitted of little or no expression. In our case there was not an appreciable involvement of the muscles of expression. The baby was bright and happy and a favorite with the nurses, because of its cheerfulness.

The physical examination was as follows: A poorly nourished female infant, with a marked pallor: The expression was bright and changing quickly. The hair was very light, dry and scanty. The circumference of the head was 17 inches; leg, 10½ inches; chest at the nipples, 14½ inches; at the costal margin, 17¼ inches; leg, 10½ inches; arm, 8¾ inches. Total length, 24 inches. The anterior fontanelle was slightly open. There were four upper incisors, well formed, and one lower. The eyes were clear, vision apparently good and there was no squint or nystagmus. The superficial lymph nodes were palpable in the axilla and groin, but not particularly enlarged. The lungs were resonant throughout, with no interscapular dulness. The heart was negative. The abdomen was decidedly distended. The percussion note everywhere was tympanitic. The liver was palpable immediately below the costal margin; the spleen was not palpable. There was a slight suggestion of a rachitic rosary, but no craniotabes or prominent bosses. The extremities were straight, with no epiphyseal enlargements. The absence of these bony changes excludes the diagnosis of rachitis. The patella reflexes were absent. But little significance can be placed on this point, however. The interpretation of tendon reflexes in infants is difficult; sometimes they are present, and then again in the same case, with the most careful effort, they cannot be elicited. Particularly is this true in cases of nutritional disorders. Therefore, it is unjustifiable, contrary to the usual belief, to place too much value on the absence of patellar reflexes in the diagnosis of amyotonia congenita.

The weakness of the musculature of the arms and legs and back was striking. She could not hold her bottle to her lips, and though there was freedom of movement of the arms and legs, there was not the power to help herself in any way. When placed in a sitting posture she fell forward, the head falling to the table and resting on the feet. She could make no effort to hold her spine erect, as other infants of similar age and similar wasting do.

Clinical Tests. Von Pirquet, negative; Wassermann, negative.

Electrical Reactions. All muscles respond to the constant current, but not actively. No reactions of degeneration.

Laboratory Reports. Urine negative.

Blood Count. The blood count was as follows:

Reds	3,980,000	
Hemoglobin	59.0	per cent.
Polymorphonuclear	42.0	"
Mononuclears	33.0	"
Transitional	3.5	"
Lymphocytes	21.0	"
Eosinophiles	1.0	"

X-Ray. Four centers of carpal ossification, four centers of tarsal ossification.

The patient was under observation six months, during which time she twice had pneumonia. Cases of amyotonia are particularly prone to pneumonia. Every effort was made to force the feeding as far as was safe, and particularly were vegetables pushed. Well-cooked spinach and orange-juice were given daily when the condition of the bowels permitted. Hypodermic administration of ferric citrate and tincture of nux vomica by mouth were the only drugs employed. Daily massage was given. She improved slightly. We then lost track of her for one and a half years, when she again came under our observation. She is now three years old, weighs twenty-five pounds and looks like a different child. Her color is good and her appetite and digestion excellent. She can sit upright, her head does not fall as before and she can use her hands and arms perfectly. She cannot stand or walk, however, and the muscles of the legs are very flabby.

Our knowledge of the pathology of the disease is still incomplete. From the study of such necropsies and muscle biopsies as have been performed several interesting facts have been discovered. Microscopically the muscles are nearly always described as pale, soft and flabby. Fatty infiltration appears to be constant in the affected muscles. The muscle fibers themselves usually show a great diminution in size and marked increase in the number of sarcolemma and muscle fiber nuclei. Hypertrophic fibers may occur. Some have laid great stress on the fact that the peripheral nerves show a deficient myelinization of the fibers. Degeneration of the nerves has been found by Spiller and occasional cord changes have been found in the anterior horns. The brain, cerebellum and medulla are only exceptionally involved. When the nerve endings have been studied there is an apparent agreement that in the affected muscles no such endings can be found, and Faber states, as his opinion, that the primary lesion of amyotonia congenita is a congenital hypoplasm of the lower motor neuron, believing that the muscular changes are secondary to this process, and that it will be found that the original fault is in a defective germ plasm of reproductively exhausted parents. The last point is borne out by the statistical study, but, as regards the congenital hypoplasm of the lower motor neuron, it seems far more justifiable to assume that this hypoplasm is secondary to the muscular changes or is due to the same causes as the muscular changes. How otherwise can the frequently reported improvement in cases be explained?

Not attempting to show the percentage of cases showing improvement, but rather that improvement and in many instances decided improvement does occur, the following cases are referred to.

Concetti: 1. Marked improvement on massage and electricity.

2. Marked improvement on electricity, massage and thyroid extract.

3. Marked improvement on thyroid, with disappearance of pseudo-edema and fat pads and improvement in mobility.

4. Improvement on massage and thyroid extract.

Reitschel: Improvement after treatment with faradic current.

Schippers: Gradual improvement after eight months.

Beling: Improvement.

Charles: Began to walk and talk at two years.

Duthoit: Improvement on thyroid extract.

Thorspechen: Improvement on exercise and faradization.

Bienfait: Improvement beginning after treatment with massage and electricity.

Gott: Improved.

Gordon: Two cases showed improvement.

Hertz: Improvement.

Reiner: Improvement was marked after two months.

Collier: Improved on rest and exercise.

Purser: Improved greatly.

Shoog: Two cases improvement.

If a congenital absence of nerve plates in the muscles does not seem likely because of this marked improvement that so often occurs in these patients, to what then can we turn as a possible etiological factor? Some interference with the normal growth of muscles is apparent. The processes of metabolism and the possible inhibitory influences must be considered. The nature of the feeding does not seem to have proved a factor. Diets that permit growth in other children do not seem to be of any avail here. All we know about the metabolism of amyotonia congenita is that (1) there is a diminution of hepatic functional activity, as manifested by the presence of acholia; (2) a normal calcium retention; (3) a low creatinin excretion, and accompanying this a relatively high creatin excretion.

The possibility of some inhibitory influence on muscle growth then seems at present to be all that remains in our process of deduction by exclusion. The hormones of the endocrin gland possess, we know, an accumulative or inhibitory influence upon the hormones of one or more of the other endocrin glands and upon metabolism.

The parathyroids belong to the inhibitory group and their action retards protein exchange, upon which muscle growth depends. Removal of the parathyroids is followed by increased muscular activity. Tetany, the laryngismus stridulus that is so often associated with it, myoclonus, paralysis agitans and mytonia may be related to a decreased activity of the parathyroids. Retarded protein exchange and the atonia may both then be ascribed to a hyperactivity of these glands. It is well known that there may be a functional disturbance of these glands without there being any apparent change in their structure. Pathologic studies are therefore not conclusive. It is interesting to note, however, that in the autopsy notes of B. S. Veeder on the case reported by Griffith and Spiller the pancreas is said to be a dark purple in color and that in the thymus there was a marked congestion of the bloodvessels.

The recorded studies in the literature do not permit any definite conclusions, and my own work on this one case is in the nature of an observation only. The subject is intensely interesting, however, and we are justified in the hope of a successful solution as our knowledge becomes broader.

A CASE OF DIABETES INSIPIDUS IN A CHILD TWO YEARS OF AGE

By PAUL B. BENDER, M.D.
ASSISTANT IN THE PEDIATRIC CLINIC

Polyuria, or diabetes insipidus, is a chronic disease characterized by persistent passage of large quantities of clear urine of low specific gravity.

It is of exceptional occurrence in infants, but less rare in older children, and is met with in boys and in girls alike. Among the exciting causes are emotions, fear, cold, some acute disease or a fall on the head. Among the predisposing causes are a neuropathic heredity, hysteria or insanity in the ancestry.

From a clinical standpoint we may classify the cases under two groups:

1. Primary or idiopathic (no evident organic basis).
2. Symptomatic (disease of brain or elsewhere).

Among the idiopathic group cases have been reported where parents have had glycosuria or albuminuria; in others, the disease has followed rapidly the copious drinking of cold water or a drinking bout, or has set in during the convalescence from an acute disease.

Among the symptomatic group cases reported have been attributed to injury or disease of the nervous system, traumatism to the head or trunk, tumors of the brain, lesions of the medulla, tumors and aneurysms in the abdomen, tuberculous peritonitis, and in carcinoma there may be excessive polyuria. Syphilitic lesions of the brain involving the basal region are particularly likely to be associated with this disease.

From comments in the *Journal of the American Medical Association*, "considerable research work has led to the conclusion that important relationship exists between this disease and disturbances of the pituitary body. As studies of pituitary disease have progressed it has been noted that polyuria is not an infrequent symptom. In dystrophia adiposogenitalis this symptom may be pronounced; in acromegaly both polyuria and glycosuria may be present, and in certain types of infantilism associated with pituitary disease the passage of large quantities of urine may be an outstanding feature. It has been noted, too, that tumors in the neighborhood of the hypophysis which exert pressure on this gland can be associated with the clinical picture of diabetes insipidus. Experimental work, such as has been done by Harvey Cushing in this country, has likewise shown a relationship between the pituitary gland and the excretion of urine. Damage to the pituitary gland during operative procedure may result in polyuria, and ablation of the gland in animals likewise results in polyuria. These observations all tend to show that there is a definite relationship between the syndrome that we call diabetes insipidus and disease of the hypophysis, particularly the posterior lobe. The importance of such a relationship has a bearing on diagnosis, and suggests the careful roentgenographic examination of the skull in cases of this nature. Theoretically it suggests the administration of pituitary extract as a curative measure, which, however, according to scientific records, has been a failure."

An interesting case admitted to the children's ward of the Episcopal Hospital, service of Dr. C. N. Sturtevant, to whom I am indebted for the privilege of reporting this case, is as follows:

Francis, aged two years, male, white. Admitted September 12, 1919. Chief complaint: Excessive thirst; anorexia, polyuria.

Family History. Mother, twenty-two years of age, brother five years, living and well.

Father, twenty-eight years. Has had polyuria for many years without any interference in health.

The mother has always enjoyed good health, except during pregnancy, especially the latter months, developed insatiable thirst, which ceased as soon as the patient was born.

History of ancestry negative for polyuria as far as could be learned.

Personal History. Child of full term; normal birth; weight, nine pounds. Bottle-fed on milk mixtures, with a history of frequent gastro-intestinal upsets, requiring more or less constant attention by family physician until eighteen months old. No history of acute illness or injury.

Recent Illness. About a year ago the mother noticed that the baby had abnormal thirst and six months later began passing copious quantities of water. As the thirst progressed, vomiting often followed. The child became very restless, with crying spells, relieved by drinking water. No other liquids would satisfy the vomiting, which frequently followed drinking, and apparently did not relieve the child's cravings, so the procedure was repeated until the water was retained. As the condition was progressive, diapers constantly wet, the child unable to eat, he was brought to the hospital.

On Admission. (Examination by Dr. Sturtevant.) Well developed baby of about two years; good color; weight twenty-two pounds.

Head: Well shaped, of round type, hair not copious, light in color, not unduly dry.

Eyes: No keratitis, pupils equal.

Ear and Nose: Negative.

Mouth: Sixteen well-shaped teeth in good condition. Tonsils enlarged.

Chest: Well shaped, normal resonance, equal expansion, no abnormal breath sounds, no rachitic rosary.

Heart: Normal outline; sounds are clear.

Abdomen: Moderately distended, spleen not palpable, liver palpable one finger's breadth below costal margin.

Extremities: Well shaped, no enlargement of epiphyses. Skin clean. Lymphatic glands not palpable.

Mentality: Child exceedingly bright. Breathing mostly through mouth when quiet. Cried vigorously throughout examination and voided urine constantly.

Observations and Findings: *Complete blood count* was normal except for a preponderance of lymphocytes.

Wassermann reaction on blood and spinal fluid was negative.

X-ray examination of sella turcica was negative.

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The child was given a salt-free diet, whereupon the chlorides in the urine diminished exactly one-half. The intake of water ranged from 3000 to 5000 c.c. and the urinary output from 1700 to 4200 c.c. in twenty-four hours. The urine was colorless, acid, of low specific gravity, and otherwise negative.

Date.	Percentage, Chlorides,	Percentage, NaCl.	Percentage, Chlorin.	Percentage, phosphates.	Percentage, P_2O_5.	Percentage, Urea.	Acetone.	Diacetic acid.	Amount output, 24 hrs.
Sept. 21	0.8	0.10	0.06	1.50	0.045	0.42	Neg.	Neg.	2950
22	1.0	0.13	0.08	1.25	0.042	0.19	“	“	3500
23	1.0	0.13	0.08	1.00	0.040	0.48	“	“	2200
24	1.0	0.13	0.08	0.80	0.030	0.18	“	“	2380
				SALT-FREE DIET					
26	0.5	0.07	0.04	1.50	0.045	0.30	Neg.	Neg.	2275
27	0.5	0.07	0.04	1.50	0.045	0.12	“	“	1700
28	0.5	0.07	0.04	1.50	0.045	0.42	“	“	2200
29	0.5	0.07	0.04	1.50	0.045	0.35	“	“	2175

The intense thirst was such that the child on several occasions had gotten out of its crib, climbed into the bath tub and with its head under the faucet attempted to satisfy his cravings for water. Dr. Mendel, resident physician, states the child has frequently taken eight cups of water without stopping, crying between each cup until more was given.

As the child was removed from the hospital before a further study could be made, he was classified as a case of diabetes insipidus of idiopathic origin.

March 20, 1920: Patient was visited at the home. The clinical picture of diabetes insipidus about the same as on admission. Mother states that the child was greatly improved by hospital treatment and remained so until recently. Expects to have the child admitted to the hospital again in the near future.

A CASE OF BACTEREMIA COMPLICATED BY SUBCUTANEOUS EMPHYSEMA OF THE FACE, NECK AND CHEST

By JOHN B. CARSON, M.D.
PHYSICIAN TO THE HOSPITAL

The following case was interesting both on account of the complication of subcutaneous emphysema and also on account of failure to locate the primary focus of infection:

Mrs. G. H., aged forty-five years. Was first seen by me on January 17, 1920, in consultation with her physician, Dr. E. F. Walsh. Chief complaint: Difficulty in breathing and difficulty in swallowing.

History of Present Illness. On January 14, following an attack of coughing, she complained of sudden, sharp, severe pain in the back of the neck. This was severe enough to make her cry out, and was ascribed to a cervical neuritis. The following day a "fulness" of the neck was noticed and pain was present in the back and in the left arm.

January 16. Swelling had greatly increased on both sides of the neck, and the eyelids on both sides were also swollen.

Dr. Walsh was then called in and found the swelling due to subcutaneous emphysema.

January 17. Patient worse. Temperature, 102°; breathing was stertorous; swallowing difficult; the swelling of the neck was more pronounced and she complained of pain in the back of the neck.

Physical examination at this time showed the patient prostrated and appearing very ill. The eyelids of the right eye were both greatly swollen and crepitation was present. The eyelids of the left eye were also swollen and also contained subcutaneous emphysema, the swelling not being as great as on the right. Both eyeballs were prominent when the lids were retracted; the pupils were equal and reacted to light and accommodation. The conjunctivæ were normal. The face was swollen equally on both sides. The mouth could only be partially opened. The fauces and tonsils were reddened and a mucopurulent material was present. There were several bad teeth. The sinuses and mastoids were negative. The neck was rigid and painful, swollen on both sides and contained much subcutaneous emphysema. Edema and tenderness were also present but no definite point of intensity could be determined and no mass was detected. The supraclavicular and infraclavicular fossæ were obliterated by the swelling. The subcutaneous emphysema extended anteriorly down to the level of the fifth rib and posteriorly to the level of the spines of the scapulæ. It seemed a trifle more marked on the right side. It did not extend into the arms. The heart was normal in size and position, the sounds were regular, rapid and of poor quality. No murmurs were heard. The pulse was rapid, regular and small. The chest was tympanitic anteriorly and posteriorly over the emphysematous area. It was impossible to elicit accurate voice and breath sounds over this area on account of the marked crepitation. Some large moist rales were heard at both bases posteriorly. Respiration was rapid, shallow and labored. The abdomen was flat, firm, and tender over the liver and spleen. The liver was normal in size and the spleen was not palpable. The knee-jerks were normal, no Kernig, no clonus and no Babinski. Temperature, 103⅖°; pulse, 120; respirations, 32. A tentative diagnosis was made of rupture of the lung with possible abscess and the patient removed to the Episcopal Hospital.

Dr. Astley P. C. Ashhurst then saw the patient and made multiple incisions in the neck, but was unable to locate any focus of infection. He thought there was mediastinitis.

January 18, A.M. Patient worse. A loud systolic murmur was heard at the apex and was transmitted to the left axilla. Pulse rapid and small. Swelling of the neck unchanged. Patient could not swallow at all and expelled enteroclysis. Temperature, 100⅘°; pulse, 108, respirations, 22. Same afternoon patient was worse; heart sounds of poorer quality; murmur less pronounced; unable to determine focus of infection. Temperature, 102⅗°; pulse, 120; respirations, 36. Same night, dyspnea and cyanosis were very marked. A tracheotomy was done, but gave only slight relief. Death occurred two hours later.

The previous medical history was not notable until two weeks before the onset of this illness, when the patient had had a "bad cold," from which she had not fully recovered.

Laboratory Findings. Culture from throat was negative for Klebs-Loeffler bacilli.

Urinalysis: Straw color; specific gravity, 1020; reaction, acid; albumin, light cloud; sugar, negative.

Microscopic: Many hyaline and granular casts; much epithelium.

Blood Culture: Positive for Staphylococcus pyogenes aureus.

Postmortem Findings. A large abscess was present, extending from the mediastinum up into the neck on each side. Necrotic peribronchial glands. Acute vegetative endocarditis. Multiple abscesses of liver and spleen. Site of rupture of pulmonary alveoli could not be determined on account of adhesions.

The subcutaneous emphysema was evidently due to rupture of pulmonary alveoli induced by coughing. The mediastinal abscess was evidently caused by necrosis of peribronchial glands. Death was due to bacteremia.

INAUGURATION OF A SPECIAL CLINIC FOR SYPHILIS

By WARREN WALKER, M.D.
SYPHILOLOGIST TO THE HOSPITAL

It is only within a very recent period that syphilis has been treated in special clinics, and even at the present time there are very few such clinics in the city of Philadelphia. Syphilis until recently has not been taken seriously enough, the cases being treated in a more or less haphazard manner in whatever clinic they first appeared: The eye lesions in the eye clinic, the skin lesions in the skin clinic, the heart lesions in the medical clinic, and so on.

To my mind the following arrangement is necessary: A chief of clinic, to whom all cases of syphilis should be referred. After examination, if a case should be found that needed special attention from the nerve, eye or medical specialist, it could be referred for examination; or a still better method would be to have a doctor from each of these clinics in attendance during the clinic hours. In this manner a complete examination could be made at the first visit and great loss of time for both physicians and patients could be avoided.

In addition to the chief of clinic there should be as many assistants as may be required to give medication, etc., depending entirely on the number of patients to be treated, one or two nurses, a social worker, who should take the social history, follow up cases and assist in keeping the records. Having a capable worker connected with the dispensary is very important, aiding especially in getting the patients to return for treatment, for we all know as soon as the subjective symptoms disappear it is difficult to convince most patients of this type that further treatment is necessary. The worker in this department should have no other duties. An orderly is likewise necessary.

The equipment necessary is as follows:

1. A waiting room.

2. A consulting and examining room in which the records of each patient are kept on file.

3. An operating room where arsphenamine in its various forms can be administered. This room should be well lighted, from above if possible. It should be equipped as follows:

Operating tables with arm rests. It is well to have the tables curtained off, thus affording a certain amount of privacy.

A small table at the head of each operating table for trays, test-tubes, needles, etc.

A large table upon which should be placed the apparatus necessary to prepare arsphenamine for injection, as follows:

Arsphenamine, files, sterile distilled water, graduated mixing jars, glass rods, funnel, sterile gauze and creton, pipettes, sodium hydroxide, hypodermic syringe, adrenalin, aromatic spirits and atropine, Luer syringes 10, 20, 30 and 50 c.c. sizes, with proper needle for administering "neo"; graduated containers, rubber tubing and connections for giving arsphenamine by the gravity method, stands to hold gravity apparatus, test-tubes, Wassermann needles and a dark field microscope.

4. A room should be provided as a recovery or rest room in case of reaction.

In conjunction with the dispensary the privilege of using the wards of the hospital for spinal puncture cases should be obtained and a certain number of beds set aside for syphilitic cases that need hospital care.

At the present time there is being inaugurated at the Episcopal Hospital a dispensary for the treatment of syphilis. It is hoped by the writer that it will eventually be as described above, as there is a very large field from which to draw cases and a great need for such a dispensary.

At the present time the clinic hours are Tuesdays and Thursdays, at 1.30 P.M., and a night clinic on Mondays at 6.30, for those patients who cannot get away from work during the day.

REPORT ON TETANUS[1]

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ABSTRACT

THE *pathogenesis* of tetanus being fairly well established, rational methods of prophylaxis and treatment are possible.

Care of the wound (*débridement*, *excision*, *extraction*) is the most important factor in *prophylaxis*, but second only to it is the prophylactic use of antitoxin. If the latter alone is available, its prompt use may avert or at least postpone and render less severe an attack of tetanus even when early proper care of the wound is impossible, as often in military surgery. The unusual frequency during the German War of forms of tetanus rarely seen in civil life (late, chronic, local and recurrent tetanus) is attributable to the general prophylactic use of antitoxin.

Treatment requires (*a*) elimination of the source of the toxin (proper care of the wound), (*b*) neutralization of the toxin already formed, (*c*) administration of drugs which depress the spinal cord, and (*d*) nursing the patient. Antitoxin should be given in large amounts *as soon as possible* after the diagnosis is made. Intravenous administration serves only to neutralize the circulating toxin, and must always be supplemented by intraspinal administration, which has been proved clinically (both in laboratory work and in patients)

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[1] Reprinted from Comptes-Rendus de la Société Internationale de Chirurgie, 1920; and from Archives of Surgery, 1820, i, 407.

to be efficient in neutralizing toxin already absorbed into the nerve roots or spinal cord.

If early and efficient treatment is administered, the mortality should not exceed 20 per cent. The author reports 17 personal cases, from civil life, with 7 deaths (41.1 per cent.). Among 12 patients whose treatment was early and efficient, there were only 2 deaths (16.6 per cent.).

A. PATHOGENESIS OF TETANUS. The following propositions may be accepted as proved:[1]

1. The disease is a pure toxemia; the bacilli or their spores may exist indefinitely in the tissues, and *no symptoms will be produced unless toxins are formed.*

It is important to realize, as pointed out by Tulloch (*Proc. Royal Society*, London, 1917–19, Series B, xc, 529–541), that at least *three different strains* of toxic B. tetani are recognized, and more than one non-toxic strain. And one must not forget the necessity for *symbiosis* of the spores of tetanus bacilli (and even of the bacilli themselves) with certain other organisms before toxin can be produced. Tulloch (*British Med. Jour.*, 1918, i, 614) found B. welchii and Vibrion septique very valuable in experimental work; while the simultaneous presence in the wound of the pyogenic organisms has long been appreciated as a predisposing factor of the greatest importance.

2. In experimental tetanus, when small animals are used, the form known as *Tetanus ascendens* occurs: here the symptoms of the disease begin in the inoculated extremity and though other neighboring parts may become affected subsequently, yet death or recovery usually occurs before trismus and retraction of the head develop. In the larger animals and in man, however, the symptoms usually begin first in the muscles of the neck and jaws, *no matter where the point of inoculation;*

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[1] The evidence on which they are based is detailed at length in a monograph by Ashhurst and John (Philadelphia): The Rational Treatment of Tetanus, Am. Jour. Med. Sc., 1913, cxlv, 806 and cxlvi, 77. (All references prior to that date may be found there.)

subsequently the muscles of the back and trunk are affected, and finally the extremities. This form of the disease is known as *Tetanus descendens*.

The explanation of this difference resides in the different methods of inoculation. In experimental animals the toxin was usually injected into muscle, came into immediate relation with the motor nerves, was absorbed rapidly by them, and the part of the spinal cord first to be affected was that in anatomical relation with the wounded limb. In man, however, the usual point of inoculation (in civil life at least) is the hand or foot, and much of the toxin enters the general circulation and is carried to the end-plates of all nerves throughout the body; and toxin which is thus carried to such short nerves as those of the facial, cervical, and spinal muscles will reach the spinal cord through these short nerves, and will begin to produce tetanic spasm in the parts supplied by these short nerves, even before the toxin in the wounded extremity has had time to ascend the nerves of that extremity as far as the anatomically related region of the spinal cord. Tetanus following wounds of muscular parts in man is not infrequently inaugurated by symptoms of local tetanus.

3. It is a fact that *the toxin ascends the peripheral nerves to the spinal cord.*

The peripheral nerves have little power of neutralizing the toxin—no more power than has the liver, for instance; and after death toxin may be recovered from the nerves as readily as from the internal organs. The peripheral portions of the nerves are found to give up more toxin than those portions nearer the spinal cord: in other words, *the nearer the toxin gets to the spinal cord the more impregnably entrenched does it become in the nerve tissue; and the readier the access of the toxin to the central nervous system, the less is the amount required to kill.*

The toxin ascends the nerves:

(*a*) By way of the axis cylinders (perhaps by means of a centripetal protoplasmic current).

(*b*) By way of the lymphatic channels of the epi- and perineurium.

What proportion of toxin ascends by each of these channels does not appear to have been determined. The researches of Robertson (*Am. Jour. Med. Sc.*, 1916, clii, 31) and of Teale and Embleton

(*Jour. Path. and Bact.*, 1919, xxiii, 50), however, indicate that the lymph channels are of more importance than was formerly thought. Robertson's experiments further indicate that the toxin gains access to the cord *through the motor roots.*

When the toxin has reached the spinal cord it spreads to neighboring parts of the cord, both to the sensory side and to the neighboring motor regions.

Tetanus dolorosus may be produced experimentally by injecting toxin into the sensory nerve roots, and the animals will die of exhaustion before motor phenomena develop.

4. *The toxin also enters the general circulation*, but only when this toxin reaches the spinal cord does it produce characteristic tetanic symptoms.

In animals rapid emaciation sometimes follows administration of tetanus toxin, when no tetanic symptoms are produced; probably these cases, described as *Tetanus sine tetano*, are due to the action of the toxin on structures other than the nervous system.

It is probable that most of the toxin which reaches the central nervous system from the general circulation does so only indirectly through absorption by peripheral nerves. Albert (*Compt.-Rend. Soc. Biol.*, Paris, 1918, lxxxi, 1127) contends that most of it is carried directly to the central nervous system by the blood; but he presents no evidence to disprove the former theory, which has recently been confirmed by the researches of Teale and Embleton (*loc. cit.*).

5. *Cause of the Symptoms of Tetanus.* The toxin stimulates the motor cells of the spinal cord, with the result that the muscles controlled by these cells are thrown into tonic spasm; the toxin also renders the sensory side of the cord extremely susceptible to external stimulus, so that very insignificant stimuli, such as the slamming of a door, jarring the patient's bed, a sudden draught of air, etc., will bring on a clonic convulsion, or at least will greatly intensify, for the moment, the tonic spasms.

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B. PROPHYLAXIS OF TETANUS.

1. *Certain classes of wounds*, received in certain surroundings, are more often followed by the development of tetanus than

are ordinary wounds. The Bacillus tetani normally infests the intestinal tract of horses and cattle (it is found in the intestinal tract of perhaps 5 per cent. of mankind) and is deposited with their dung. Therefore wounds contaminated with barnyard or highly cultivated garden soil, those produced by dragging in street dust, etc., are especially liable to be infected with tetanus bacilli. Gunshot wounds are liable to contamination not *per se*, but only as these conditions, and others, presently to be mentioned, obtain.

Growth of the organisms is favored by *anaërobic conditions of the wound.* These are present in any wound in which there is *tissue destruction*, which implies cessation of circulation in the devitalized tissues; the best culture medium for tetanus bacilli is that which contains some dead organic tissue. *Contused, lacerated* and *gunshot wounds* offer ideal conditions for the development of any bacilli present; and as the bacilli are carried into the wound only by a missile (shell fragment, fragment of clothing, splinter of wood, rock, etc.) and usually remain attached to this missile, it follows that *wounds with retained missiles* offer the most favorable conditions possible for the development of tetanus. Punctured wounds are to be dreaded not because the vulnerating instrument is retained, which it seldom is, but because it was contaminated either (*a*) even before it pierced the skin, or (*b*) because it carried infection from the skin or clothing deeper into the tissues. Even a superficial brush-burn may give rise to tetanus (as in a case in my own experience) provided the skin or the vulnerating surface carries tetanus bacilli.

2. *Care of the wound* is the first step in the prophylaxis of tetanus. It is my own firm belief that *efficient care of the wound as soon as possible after its receipt is by all means the most important feature in prophylaxis.*[1]

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[1] The report by Clark (Boston Med. and Surg. Jour., 1917, clxxvi, 541) of 100 consecutive cases of punctured wounds of the foot, produced by nails, without a single bad result, and the fact that no tetanus antitoxin was employed, indicates the efficiency of prompt and proper care of the wound.

This includes:

(*a*) *Mechanical cleansing* (*débridement; extraction* of foreign substances; *excision* of devitalized tissue), and

(*b*) *Chemical disinfection* (3 per cent. alcoholic solution of iodin remains, in my own opinion, the best agent for this purpose; certainly no cauterizing agent should be used, as this will produce more dead tissue).

3. *Prophylactic use of antitoxin* holds second place, but second only to care of the wound.

The fact that antitoxin is of use as a prophylactic injection scarcely needs discussion. Accumulated experience during the German War merely served to emphasize its value as already ascertained in civil life. In the War of the Rebellion there occurred, according to Sanford (*Internat. Assn. Med. Museums Bulletin*, 1918, No. 7, 365) 505 cases of tetanus among 246,172 wounded, or a little over 2 per 1000. In the American Expeditionary Forces, during the German War, according to figures furnished to me by the Office of the Surgeon-General, U. S. Army, there developed 36 cases of tetanus among 223,067 wounded, or less than 2 per 10,000. Even allowing for the vast improvement in the mechanical and chemical disinfection of the wounds, such a remarkable diminution in the incidence of tetanus could never have been secured without antitoxin, especially in view of the highly contaminated nature of the soil in which much of the fighting was done.

Reference may further be made to the communications of Bazy (*Bull. Soc. Chir.*, Paris, 1916, xlii, 2919) who observed 200 wounded from the same sector, of whom 100 received serum when wounded and only one developed tetanus (the day after the injection), while the remaining 100 received no serum, with the occurrence of 18 cases of tetanus; of Columbino (*Bull. Soc. Chir.*, Paris, 1916, xlii, 2913), who narrates that among 200 wounded received in his service, all but 2 had received a prophylactic injection of serum, and only these 2 developed tetanus; of Walther (*Bull. Soc. Chir.*, Paris, 1917, xliii, 71) whose service received 270 wounded German prisoners, to as many as possible of whom antitoxin was given, with the result that of the whole number 19 developed tetanus, only one of these cases being in a patient who had received the serum (and in his case the disease appeared the day after the injection); of Fredet (*Bull. Soc. Chir.*, Paris, 1917, xlii, 1338) who observed 12 wounded German prisoners (to whom the German surgeon left in charge refused to administer proffered French antitoxin, because he believed it to be of no value) 6 of whom developed tetanus, while among a larger number of serum-treated French (wounded in the same battle, and in an adjoining ward) not one developed tetanus. And especially would I call attention to that graphic representation of the prophylactic value of antitoxin published in the *Arch. Méd. Belges* (1917, lxx, 834) which gives a curve constructed from Sir David Bruce's reports in the *British Medical Journal*, of the incidence of tetanus in the British Army, indicating an incidence of from 15 to 32 cases of tetanus per thousand wounded until tetanus antitoxin was administered as a matter of routine to all the wounded, whereupon the incidence fell to 2 or 3 per thousand and never subsequently exceeded these figures. Nor against these facts can it be argued that tetanus was prevalent only at the first Battle of the Marne; for as Leriche (*Bull. Soc. Chir.*, Paris, 1917, xliii 76) points out, it was prevalent also in other sectors wherever serum was not available for prophylaxis, and it complicated even clean perforating bullet wounds which neither then nor at any other period of the war were treated by operative disinfection; yet in these wounds as well as in wounds which received operative treatment the incidence of tetanus became negligible whenever serum was available. The fact that the wounded of the British Army when cut off in Kut-el-Amara began to develop cases of tetanus, but that the incidence fell at once to its previous low figure when an aviator arrived with a supply of antitoxin and its prophylactic use was resumed (*Arch. Méd. Belges*, 1917, lxx, 834)—this, I say, is but another proof of the prophylactic value of antitoxin.

There are three factors to be considered in connection with the prophylactic use of the antitoxin: (*a*) The quantity to be administered; (*b*) the site of the injection; and (*c*) the frequency with which it should be administered.

(*a*) *The Quantity of the Injection.* The usual prophylactic dose is 1500 units. U. S. A.

The United States Government's standard method for testing the strength of tetanus antitoxin is as follows: "The immunity unit for measuring the strength of tetanus antitoxin shall be ten times the least quantity of antitetanic serum necessary to save the life of a 350-gram guinea-pig for ninety-six hours against the official test dose of the standard toxin furnished by the Hygienic Laboratory of the Public Health Service." The official test dose of toxin is one hundred times the smallest quantity of toxin which will kill a guinea-pig within ninety-six hours.

Taking the average weight of persons injected as 150 pounds (70 kilograms), and calculating the potency of the antitoxin from the known value of a unit (U. S. A.), it is evident that 1500 units is a little over two thousand times the minimal lethal dose of toxin in such an individual. Such an amount is by no means excessive, in view of the fact that the amount of antitoxin required to prevent death increases in geometrical progression with the lapse of time. After the lapse of one hour, twenty four times as much antitoxin is required as when antitoxin and toxin are injected simultaneously (Remertz, 1911).

(*b*) *The Site of the Injection.* Usually it is administered subcutaneously. It is better, however, to administer it intramuscularly, in the immediate vicinity of the wound, in order to flood these tissues with antitoxin even before the absorption of toxin has begun. The antitoxin so injected finds readier access, it is believed, to the nerves of the wounded part, and is admitted to the circulation no less rapidly than when administered by the subcutaneous route.

(*c*) *The Frequency of the Injection.* The *first injection* should be given as soon as possible after the receipt of the wound. In military surgery the first injection almost always can be given some hours before proper care of the wound can be instituted. This fact is fortunate for the wounded man because of the frequency with which operation must be delayed; but in neither military nor civil life does this in any way impair the doctrine that proper care of the wound is the more important of the two factors in the prevention of tetanus.

It is a well ascertained fact that the antitoxin is all eliminated from the system in about eight or ten days after the injection. Its quantity remains practically undiminished for a week, and then rapidly diminishes to nothing in the course of two days. As the tetanus bacilli, if any are lodged in the wound, may not begin to produce any toxin until at least as long a time as this has elapsed, it is manifestly important that some antitoxin should be present in the system as long as the probability of the presence of toxin persists. In many cases in civil life (vaccination wounds, for instance), tetanus bacilli do not enter the wound at the time of its receipt, but are introduced secondarily by the carelessness of the patient in neglecting to return for dressings and in attempting to care for the wound in septic surroundings at home. But even if present in the wound from the first, the bacilli may lie dormant for weeks in the form of spores, before development occurs with the production of toxin.

The necessity for a *second injection* of antitoxin about the eighth or tenth day is thus very evident, and I have come to agree with Solieri (1910) who insisted that this injection must be renewed every eight or ten days thereafter until all necrotic tissues are removed and there is a clean granulating surface. The dose should be the same in these later injections as in the first.[1]

That tetanus may develop after the prophylactic use of antitoxin cannot be denied; but such cases rarely develop very soon after the injury, and when they do appear seem to be less severe than are most cases in which no serum has been administered.

Speed (*Med. and Surg.*, St. Louis, 1917, i, 14) working in a British Base Hospital, observed 6 such cases among 12,000 wounded; three of the patients recovered.

Mathieu (*Arch de méd. et pharm. navales*, Paris, 1918, cvi, 467) noted that tetanus which developed after the prophylactic use of serum gave a mortality of 26.6 per cent., while the mortality was 58.1 per cent. among those who had not received antitoxin; while Gessner and Adiger (*New Orleans Med. and Surg. Jour.*,

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[1] Vallée and Bazy (Compt.-Rend. Acad. des Sc., Paris, 1917, clxiv, 1019) have pointed out that the organism utilizes subsequent doses less and less the greater the time that has elapsed since the first dose; perhaps because it in time develops an anti-antitoxin. So that even if the supply of toxin gradually becomes less as the time elapsed since injury increases, the dose of antitoxin must be maintained at the original amount, or even be increased. The researches of these writers indicate that it may become possible to produce active immunity against tetanus by vaccination with iodized toxin; and that this active immunity may be of more value in cases of late or chronic tetanus than the progressively diminishing strength of passive immunity which is conferred by the serum.

1916, lxix, 91) report that among 11 patients who developed tetanus in spite of the prophylactic use of antitoxin (only one injection having been given in each case), there were only four deaths, a mortality of 36.3 per cent.; which is to be compared with the general mortality in the same hospital, which has varied from 64 to 78 per cent. since the introduction of serum treatment. These figures indicate that even a single prophylactic injection tends to reduce the mortality.

These early post-serum cases, a class which our French colleagues have happily named "tetanos post-serique precoce," are certainly rarer than the late post-serum cases ("tetanos post-serique tardif"); and it is undoubtedly due to the nearly universal employment of serum prophylactically that we must attribute the relative frequency, during the German War, of forms of tetanus rarely encountered in civil life: I mean the *late*, the *local*, the *recurrent*, and the *chronic* forms of the disease.

A distinction should be made between *late tetanus* (that in which no primary attack occurs under four weeks after injury); *local tetanus* (which corresponds to the experimental form known as *tetanus ascendens*, except that it never becomes general); *chronic tetanus* (that which is of long duration, irrespective of other factors; which usually is relatively mild; and which sometimes leaves contractures as a legacy); and *recurrent tetanus* (in which a primary attack is followed by others). The incubation period scarcely ever exceeds four weeks; and in late or recurrent cases it should be dated from the arousing injury or operation, not from the original wound (Hesse: *Deutsch Arch. f. klin. Med.*, 1917, cxxiv, 284). Speed (*Med. and Surg.*, St. Louis, 1918, ii, 499) has recorded a remarkable instance of recurrent tetanus: a soldier wounded in August, 1916; his wound healed without operation; he received three doses of antitoxin in the first ten days after injury. Chronic tetanus developed in October 1916; recovery was complete by February, 1917. Recurrence was noted in January 1918, there having been no new wound and no operation since the original injury. A skiagraph at this time showed a retained foreign body.

This leads to a discussion of the prophylactic use of serum before late operations on parts which have been wounded in war or in other circumstances where contamination with tetanus bacilli is feared. This subject was very fully discussed in 1917 by the Society of Surgery of Paris. As Fredet (*Bull. Soc. Chir.*, Paris, 1917, xliii, 1108) points out, it involves answers to three questions: 1. *What is the frequency and gravity of cases of tetanus occurring late, in spite of early prophylactic use of serum at the time of injury?* 2. *What is the frequency, nature, and gravity of accidents due to reinjections?* 3. *What is the efficacy of reinjections in preventing late tetanus?*

1. To the first of these questions surgeons have replied by submitting long lists of operations on such wounds with no or at most with very few occurrences of tetanus; and the cases that have occurred usually were mild. Fredet himself had only one patient develop tetanus (a mild case with recovery) among about 1000 who underwent late operation, none of these patients having received a reinjection at the time of operation. But we cannot ignore the significance of certain facts to the contrary, which will be cited below in the discussion of Fredet's third question.

2. It is an accepted fact that accidents ("serum sickness," anaphylaxis) due to reinjections are neither frequent nor serious. Cases of death from anaphylaxis are almost if not quite unknown. The fatal case related by Salvatore (*Gazz. d. Osp.*, 1917, xxxviii, 5) as one due to anaphylaxis may as readily be attributed to sepsis (local tetanus in case of undrained wound of elbow-joint with retained missile). Accidents such as erythema, urticaria, and even articular effusions, undoubtedly are regrettable, and are an annoyance to the patient; but they just as certainly are less annoying than would be the outbreak of tetanus. Moreover, according to certain observations, it is probable that the antitoxin, if administered at the time of operation *while the patient is under a general anesthetic*, will produce no anaphylactic shock. Acting upon the suggestion of Major James F. Corbett, M.C., U. S. Army (who had found it impossible to produce anaphylaxis in experimental animals which under other circumstances

were very susceptible to repeated doses of antitoxin), this practice was inaugurated by me while chief of the surgical service at the Walter Reed General Hospital, Washington, D. C., and a prophylactic injection of antitoxin was administered subcutaneously to each patient while he was under the influence of the general anesthetic at the time of any operation on parts which had been wounded in a manner to suggest the possibility of their contamination with tetanus bacilli. No anaphylactic shock was observed in any case. I learned later that precisely the same conclusion as was reached by Major Corbett, had been previously reported by Dean (*Lancet*, 1917, i, 673) who even with intravenous injections administered to 14 anesthetized patients (already having received serum previously) saw no serious results—one death occurred during the operation, but there was no proof it was due to anaphylaxis; a second patient on coming out of the anesthetic had slight shock, urticaria, and edema of the eyelids; four others had urticaria; and a seventh had edema of the eyelids alone.

3. To the question as to the efficacy of reinjections in preventing late tetanus, very significant if not conclusive facts may be submitted in answer. I may mention particularly the experience of Lt. Col. John F. Connors, M.C., U. S. Army, who during the war was Chief of the Surgical Service in the Hospital Center, A. E. F., at Savenay, while I was myself Group Consultant in Surgery. Col. Connors writes me that among 48,000 patients who passed through this Hospital Center only two developed tetanus. The rule was to administer an injection of antitetanic serum to every patient who was to have an operation on parts which had been wounded. The first patient who developed tetanus had received serum at the time of injury, but six weeks later, when Dr. Connors removed a large foreign body from his lung, *by some oversight he did not receive another injection of antitetanic serum;* he developed symptoms of tetanus within twelve hours following operation, and in spite of active treatment death occurred in thirty-six hours. In the second case,

the patient, with a compound comminuted fracture of both bones of the right forearm from gunshot wound, had likewise received antitoxin at the time of injury; but as the operation done at the Base consisted merely in manipulation of the fragments and application of a splint, *no reinjection of antitetanic serum was given;* twenty-four hours later symptoms of tetanus developed, but fortunately after a week of heroic treatment recovery ensued. Col. James P. Hutchinson, who was in command of the A. R. C. Military Hospital No. 1, at Neuilly-sur-Seine, also told me that though it was the rule to administer a reinjection to every patient at the time of a late operation on parts which had been wounded, yet it was found, in the case of two patients who developed tetanus, that *this injection by some oversight had not been given.*

So that, until its uselessness is proved, I believe it is incumbent on surgeons to administer a reinjection of serum to such patients at the time of late operations on parts which have been wounded, and especially if there is a retained foreign body or a dense cicatrix rendering all the more likely the continued but latent existence of tetanus bacilli or their spores.

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C. TREATMENT OF TETANUS.[1] The indications are (1) *to remove the source which supplies the toxin;* (2) *to neutralize the toxin already formed;* (3) *to depress the functions of the spinal cord;* and (4) *to nurse the patient.*

* * *

I. *Removal of the Source Which Supplies the Toxin.* If this source is known it should be attacked directly: the wound should be widely opened and mechanically cleansed of foreign bodies, sloughs, etc. Then it should be treated with antiseptics, and I believe a 3 per cent. alcoholic solution of iodin is the best. The wound should then be filled loosely with gauze soaked in the iodin solution. I believe any caustic should be avoided, as favoring the growth of tetanus bacilli by the formation of sloughs. If the nature of the case demands it for other reasons, amputation should be done; then the stump should be left open and treated as the original wound. Probably in many cases it will be well to follow Porter and Richardson's suggestion (1909) to excise the related lymph-nodes, particularly if they are palpably enlarged.

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[1] The importance of recognizing the disease promptly can never be overemphasized. *Premonitory symptoms must be recognized, and heroic treatment instituted without an hour's delay.*

In the case of a firmly healed wound, on the other hand, it probably will prove more detrimental to the patient to undertake any formal operation than to leave *in situ* deeply placed and apparently well encapsulated foreign bodies.

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II. *To neutralize the toxin* the best remedy is antitoxin.

As it may possibly still be thought by some that antitoxin is of no value in treatment, I can only recommend to these doubters to do what Delbet (1917) himself formerly a doubter, advised, namely to *study the subject.* To any one with an open mind, I believe the evidence though circumstantial and not perhaps positive will nevertheless seem convincing. Among many other statistics, both from civil and military experience, which could be cited, the following may suffice:

Mathieu (*Arch. de méd. et pharm. navales*, Paris, 1918, cvi, 467) notes that 960 cases under serum treatment gave a mortality of 38.8 per cent.; while 40 cases not under serum treatment gave a mortality of 80 per cent.

Dutertre (*Le Tetanos et son Traitement en Allemagne* (1914–1915), Paris 1915) quotes Permin (*Mitth. a.d. Grenzgeb. d. Med. u. Chir.*, 1913, xxvii, 1), who analyzed 330 cases of tetanus observed before the onset of the war: the general mortality was 62.1 per cent.; among those receiving serum treatment, it was 57.7 per cent.; among those receiving no serum, it was 78.9 per cent.

Other substances which are supposed to have an antitoxic effect (cholesterin, Almagia and Mendes, 1908; urea, Sewaki, 1910) have not proved themselves of value. The same may perhaps be said of carbolic acid, according to the method of Bacelli (1893), which, though employed widely and with apparent success in Italy, appears never to have given similar results in other countries. Camus (1912) showed experimentally that it had no influence on the evolution of tetanus, no matter in what amounts nor at what stage of the disease it was administered; he found that while it may possibly have some antibacterial action, it has no effect on the fixed toxin nor on the toxin in the course of fixation. *Antitoxin,* alone, injected simultaneously into the cerebrospinal fluid by lumbar puncture, intravenously, and subcutaneously, *gave very much better results.*

Exactly how the antitoxin acts upon the toxin is not known. Krauss and Amiradzibi (1910) from their numerous experiments, concluded that it draws the toxin out of the cells where it is lodged, but does not itself enter into the toxin-containing cells. Recovery under the use of antitoxin they think depends on the possibility of this diffusion, and they believe that this occurs in the human body, it being unnecessary for antitoxin to be in direct contact with toxin for it to exert its neutralizing influence on the latter.

But here, again, as in the question of its prophylactic use, we must inquire as to the quantity, the site and the frequency, of the injection.

1. *Quantity of Antitoxin Injected.* No matter what the method of injection, *the most important thing is to get the maximum quantity of antitoxin indicated into the patient's body as soon as possible.* Delay even of a few hours may determine a fatal result; 15,000 units given within the first three hours after symptoms develop are of more use than 50,000 units given after six hours, or given in divided doses. It should be made a rule *to administer the total quantity indicated as nearly as may be all at one time;* and after this overwhelming dose of antitoxin has once been given, to keep the patient's system supplied with antitoxin, though in moderate amount, until his recovery seems assured. Dean (*Lancet,* 1917, i, 673) found the physiological action of antitoxin in the blood serum could still be demonstrated as long as twenty, thirty, and even thirty-nine days after a single intravenous injection (30,000 units, U. S. A.).

As will be pointed out below, if the antitoxin is administered *subcutaneously*, immense quantities are indicated. For an adult, with the usual type of case, at least 100,000 units are required in the first twenty-four hours; although a less amount may be sufficient for a child or for a comparatively mild case, one cannot be sure of the fact, and it is better to give too much than not enough. Administered *intravenously*, a less amount is sufficient; probably 15,000 to 25,000 units should be administered at first, and if no effect is apparent, or if the good effect wears off, a similar amount should be given after the lapse of eighteen to twenty-four hours. If injected *intraspinally*, from 3000 to 10,000 units should be given, according to the weight of the patient; this injection need not, as a rule, be repeated in less than eighteen to twenty-four hours. Even when administered intraspinally a certain interval must elapse before the effect of the antitoxin can be apparent. *Intraneural* injections, rarely used at present, should be made in as great amounts as the nerves will absorb. I have injected 1500 units into the sciatic nerve all at one time, on several occasions, and 750 units into each of the anterior crural and obturator nerves. If the injections are slowly made all this quantity can be introduced among the nerve fibers.

2. *Site of Injection of Antitoxin.* The following sites of injection deserve consideration: (*a*) Subcutaneous, (*b*) intraneural, (*c*) intravenous, (*d*) intraspinal.

(*a*) *Subcutaneous Injections.* Thus administered, the antitoxin is absorbed by the lymphatics, transported to the veins, passes through the lungs, and finally is distributed through the arterial system to all parts of the body. Only an infinitesimal amount ultimately reaches the motor nerves through which the toxin is being carried to the spinal cord, while the greatest part is distributed to the viscera where it is of no use. This method of administration is inferior to the intravenous in the certainty and rapidity of neutralizing circulating toxin, and since overwhelming amounts are required to produce any effect it is evidently the height of extravagance so to employ it. Should it be used, at least 100,000 units U. S. A. should be given in the first twenty-four hours. I have cognizance of two cases in the Episcopal Hospital, the cost of the treatment of which may well be compared: In the first patient antitoxin was administered subcutaneously in appropriate doses (99,000 units the first day; 65,000 units the second day; 60,000 units the third day), to a total amount of 224,000 units, and recovery followed. The cost to the Episcopal Hospital was about $180. Shortly before this I had under my own care a severer case of tetanus: on the first day 3000 units of antitoxin were administered intraspinally; on the second day 750 units were injected into the sciatic nerve, and 750 units deeply into the tissues around the wound. The patient recovered, and the total cost to the Hospital for antitoxin was about $3. The quantities of antitoxin used in each case were logically correct, but only in the second case was the mode of administration rational.

(*b*) *Intraneural Injections.* Since the more general adoption of intraspinal injections, the intraneural method has been less used. As it is a well ascertained fact that most, possibly all, of the toxin reaches the spinal cord only by travelling up its nerves, it is theoretically logical to inject the antitoxin into the nerves in order that it may not only block the nerves against further absorption but may reach the spinal cord by the easiest road. That it will reach the cord admits of no doubt (Sawamura, 1909). Accordingly it should be injected into the nerves at the roots of the limbs.

The conclusions reached by Teale and Embleton (*Jour. Path. and Bact.*, 1919, xxii, 50) do not seem justified by the proof they offer. They claim that antitoxin does not reach the central nervous system by way of the axis cylinders or neural lymphatic channels any more than it does by the arteriovenous circulation. But these latter conclusions are based largely on their inability (1) to demonstrate by sensitization tests on susceptible animals the presence of horse-serum in the spinal cord after it had been injected into the nerves; and (2) to demonstrate by inoculation of antitoxin into one sciatic nerve the physiological manifestations of its presence in the lumbar cord of an animal into whose other sciatic nerve toxin had previously been injected. Now it seems not impossible that antitoxin, like toxin, may become fixed in the cord, while it may still be recoverable from the nerves.

But as it is manifestly impracticable to expose and inject antitoxin into all of the nerves throughout the body through which toxin is being absorbed, and as it is *extremely probable* even if not categorically proved that antitoxin when injected intraspinally acts upon the toxin already in the nerve roots or spinal cord, it is nearly everywhere admitted that intraspinal are of more value than intraneural injections; especially valuable when the site of inoculation with tetanus bacilli is doubtful or unknown. The only methods we possess for reaching all the nerves at once are (1) intravenous injections and (2) intraspinal injections. In no case, therefore, should we depend on intraneural injections alone.[1]

(*c*) *Intravenous Injections.* The effects of these were studied experimentally by von Graff (1912), and subsequently by numerous other investigators. It is the surest and quickest way to neutralize the circulating toxin, and thus to prevent more of it from reaching the nerves and spinal cord; but it does not enable antitoxin to overtake and neutralize toxin already in the nerves or spinal cord, and it is the latter toxin, not the circulating toxin, which is doing the damage.

Teale and Embleton (*Jour. Path. and Bact.*, 1919, xxii, 50) came to the conclusion as the result of their experiments that antitoxin does not pass to the central nervous system by way of the bloodvessels, but that it acts simply by combining with the circulating toxin and thus prevents it from reaching the central nervous system. It is true that Dean (*Lancet*, 1917, i, 673) proved that antitoxin may be found in the cerebrospinal fluid in varying but never very large amounts, after the intravenous administration of large amounts; but if it is of any value in the cerebrospinal fluid it certainly is more rational to insert it directly by lumbar puncture, in concentrated form, than to administer it intravenously.

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[1] Pratt (New York Med. Jour., 1918, cvii, 737) adopted in one case injection (15,000 units) into the vertebral artery; one artery, he claims, delivers blood to both sides of the body. The result is not given. He points out that the circulation from this artery goes chiefly to the medulla and cord, very little to the brain. Cocain injected into the common carotid anesthetized the head and neck without medullary involvement; so he assumes that the circulation through the circle of Willis is not free in either direction.

(*d*) *Intraspinal Injections.* First used successfully in 1899 by von Leyden, who had no doubt that antitoxin was conveyed rapidly to the medullary cells after its injection into the subdural space of the lumbar cord, it is a method which is in danger of being neglected owing to modern experimental researches. But in spite of the large majority of experiments which at first sight tend to show that the intraspinal administration of antitoxin is of no therapeutic value, because it cannot be absorbed from the cerebrospinal fluid; there is sufficient clinical evidence on record to show that antitoxin can be and is absorbed into the nerve roots or cord directly from the cerebrospinal fluid. And even if this were not so, even if the *modus operandi* of antitoxin thus administered remained undetermined, that fact should in no wise hinder any rational physician from employing it by this route in the treatment of tetanus, since the clinical evidences of its value are overwhelming and irrefutable. Who does not know, except perhaps laboratory workers themselves, that the proper function of *science* is to explain *art*, not to forbid it to employ means clinically successful because so far inexplicable by laboratory researches? The facts remain, let them speak for themselves; they are both experimental and clinical; and they demonstrate beyond the shadow of a doubt that it is absolutely unjustifiable to neglect this route of administration of antitoxin in the treatment of tetanus. In addition to the now classical experimental work of Park and Nicoll (*Jour. Am. Med. Assn.*, 1914, ii, 235), we have the later researches by Sherrington, presented by Bruce (*Lancet*, 1917, i, 681) and by Andrewes (*ibid.*, 682); as well as those of Golla (*Lancet*, 1917, i, 686)—all of which demonstrate that if administered intraspinally antitoxin is effective. *How* it acts, may not yet be certainly known; let the laboratory workers continue their experiments until they find this out; that is their true function; but let them not dare to forbid its employment because its means of action remain unknown: *ne sutor supra crepidam.* It is clinical work which, here, as elsewhere in medicine, leads the way by *Art*, leaving the *how* and the *why* to be explained if possible by *Science.*

The clinical results of the intraspinal use of antitoxin are these:

RESULTS OF TREATMENT BY ANTITOXIN INTRASPINALLY.

Author.	Patients.	Recovered.	Died.	Mortality.
Luckett, 1904 . . .	4	4	0	0
Rogers, 1905	7	4	3	43.0
Hofmann, 1907 . . .	16	14	2	12.5
Permin, 1914 . . .	28	11	17	60.7
Nicoll, 1915 (collected cases)	20	16	4	20.0
Gibson, 1916 . . .	4	4	0	0
Ashhurst, 1920 . . .	14	9[1]	5	35.7

It is true that in most of these patients antitoxin was given also by other routes besides the intraspinal (*i. e.*, intravenously and subcutaneously), and that other proper methods of treatment were not neglected (use of sedatives, careful nursing, etc.); for tetanus is a terrible disease and must be fought with every available weapon. It must also, however, be borne in mind that the above list includes (at least in the case of the present writer's statistics) patients to whom antitoxin was given intraspinally so late that it is scarcely fair to include them in seeking to determine the value of this avenue of administration. If treatment is both efficient and early the mortality from acute tetanus probably should not exceed 20 per cent.

3. *Frequency of the Injections.* If the rule I have already enunciated be followed, namely to administer the total

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[1] One patient died subsequently of pneumonia.

quantity indicated as near as may be all at one time, and particularly if the intraspinal (3000 to 15,000 units) and intravenous (20,000 to 30,000 units) methods are employed, the injections will not need to be repeated very frequently. Intraspinal injections usually are to be repeated every twenty-four to thirty-six hours unless improvement commences; the intravenous injection need not be repeated for several days if improvement commences, but if the patient continues to get worse, and certainly if a less amount than 20,000 units has been injected at first, this amount should be repeated within twenty-four to thirty-six hours.

* * *

Finally, a word may be said as to the actual technique of these injections. In giving *intraspinal injections*, for which narcosis (preferably by chloroform) usually is requisite, from 5 to 10 c.c. of spinal fluid should first be withdrawn. Then the antitoxin is slowly injected, *undiluted*. I have seen no harm arise from the injection of the undiluted serum, and believe it is desirable to have it present in the cerebrospinal fluid in as concentrated form as possible. It is true that at subsequent punctures on the same patient one often finds the cerebrospinal fluid turbid or cloudy (an evidence of amicrobic meningitis) but this seems to do the patient no harm. In some cases, also, I have seen delirium follow the injection; but as the patients recovered this must be regarded as a negligible complication.

For *intravenous injections* it is customary to dilute the serum with saline solution up to a total quantity of 500 c.c. or thereabouts; and I am convinced that the injection of the undiluted serum intravenously would be injudicious because of the likelihood of producing thrombosis and perhaps embolism.

* * *

III. The third indication in the treatment of tetanus is *to depress the functions of the spinal cord.* This is equally important with the effort to eliminate the supply of toxin, and with those to neutralize the toxin already formed, because in almost every case there is a large amount of toxin which has become impregnably entrenched in the central nervous system, particularly in the spinal cord, and *none of the methods of treatment hitherto discussed has any influence over it.* Until its action is exhausted it continues to stimulate the motor and to a less degree the sensory tracts of the spinal cord, and kills the patient by exhaustion.

We have at our disposal a number of drugs whose main therapeutic action is to render the spinal cord less susceptible to stimulus; and administration of one or more of these remedies forms an integral part of any rational plan for the treatment of tetanus. The drugs most often employed are chloral, chloretone, and similar products; the bromides; magnesium sulphate; and of later years the persulphate of sodium. These drugs are to be administered until the therapeutic effect which is desired has been obtained.

Ordinary doses are not sufficient, but it is quite possible to kill the patient by an overdose. In 9 patients in my own series of cases the condition at death was noted: only 3 patients died in spasm or convulsion, while 6 died in complete relaxation; and in some of these cases the condition was due to overdoses of the spinal depressants employed. Especially dangerous I believe is magnesium sulphate, which as Bérard and Lumière (*Presse Méd.*, 1918, xxvi, 469) express it, is efficient only in "*doses paramortelles.*" I agree with them also that chloral in doses of 4 to 10 grams daily is the most efficient and inoffensive of spinal depressants; and I habitually employ it in conjunction with the bromides.

Their own experience with intravenous injections of persulphate of sodium (Lumière: *Lyon Chir.*, 1915, xii, 411) is worthy of great attention. Lumière employed it in 8 isolated cases before the war, only 2 of the patients dying; and during the war he states that of 29 patients treated efficiently by this method, only 13 died, a mortality of 45 per cent. He injects intravenously once or twice daily 20 c.c. of a 5 per cent. solution of the drug, which should be *pure* and *neutral* in reaction. These injections are to be repeated daily or less often for the first or second week of the disease. But as Bérard and Lumière (*Presse Méd.*, 1918, xxvi, 469) themselves acknowledge, it is not so efficient in very acute cases, and chloral should be used also. They report a total of 116 cases personally treated during the war, with 58 deaths, a mortality of 50 per cent. But it must be remembered that many of the war cases of tetanus were late, chronic, local or recurrent cases, and so the mortality cannot well be compared with that of the cases seen in civil life. The persulphate of sodium should be preserved in solid form (dry) in 5 gram quantities, in sealed tubes. The *solutions* can be preserved only a few days (not more than eight) and are decomposed by heat. They must be prepared freshly by dissolving the contents of the 5 gram tube in 100 c.c. of cold sterile distilled water. The solution corrodes metals, and metal syringes and needles must be very carefully washed after use. After the injection the clonic spasms cease, but the tonic spasm is unaffected. Vomiting occurs in about half the cases after the injection. He regards this remedy as antitoxic as well as depressant, and seems not to have employed any antitoxin in addition. Other authorities, however, doubt its antitoxic power.

Spinal anesthesia is suggested by Bérard and Lumière as a remedy for obstinate constipation; and in one patient they secured marvellous relief from respiratory cramps by injecting a local anesthetic into both phrenic nerves at the root of the neck.

* * *

IV. The patient, as well as the disease, must be treated; but it is perhaps unnecessary here to dwell further upon nursing, feeding, and meeting every untoward symptom as it arises.

I have had under my personal observation 18 cases of tetanus, all in civil life; 7 of these patients died (38.8 per cent.), one of them (Case 13 in the annexed table) from pneumonia, after apparent recovery from the tetanus. In 3 other fatal cases treatment was neither early nor efficient enough to be successful (Cases 6, 9, 11); while the death of one patient (Case 10) was clearly due to an overdose of magnesium sulphate intraspinally. This leaves 13 cases in which treatment may be regarded as early and efficient; among these, only 2 patients died, a mortality of 15.38 per cent.

In conclusion, I may summarize my views as to the *treatment* of tetanus by narrating the history of the next to the last patient to come under my care; he was treated early and efficiently, and recovered.

CASE 17.—M. H. J., thirty-two years of age, a member of one of the Faculties in the University of Pennsylvania, referred to me by Dr. Alfred Stengel, was seen in my office about 7 P.M., November 26, 1916. He stated that two days before (November 24, 1916) he noted in the morning that his cheeks felt stiff, as if they had been out in the wind all day (which they had not). The next day he felt the same way, and on the morning of the 26th he noticed in addition to the sensation of stiffness in his cheeks and jaw that he was stiff in the neck and shoulders, as if from overexercise. Then only did he recollect that on November 16 or 17, while doing some carpenter work in his garden, he had stepped on a rusty nail, which had perforated his shoe and penetrated his foot for 2.5 to 3 cm. He had extracted the nail, squeezed out a few drops of blood and the puncture had given no further trouble, and he had forgotten about it until the persistence of stiffness in his jaws and neck made him apprehensive of the development of tetanus.

Examination was negative, except for slight stiffness in the jaws, but the mouth could be opened well. Nevertheless, his sensation of stiffness was pronounced in jaws, neck and shoulders.

I took him with me at once to the Episcopal Hospital, and riding beside him in the car felt anxiety lest he should suddenly grow worse and have a convulsion. There seemed no doubt about the diagnosis; if delay were allowed, the treatment, no matter how energetic, would come too late to be efficient.

By 8 P.M. (one hour after he came to my notice) this patient had received, under local anesthesia, 10,000 units of concentrated antitoxin intraspinally by lumbar puncture. (The cerebrospinal fluid withdrawn was reported (Dr. C. Y. White) as containing no cells; injected into a mouse, it had no deleterious effect.) The puncture in his foot could not be certainly identified and nothing was done to it. He received also, by mouth, chloral hydrate, 1 gm., and sodium bromide, 2 gm. By 12 midnight his temperature had risen to 39° C., but fell again to 37.8° C. by morning.

November 27, 1916. The patient is delirious, muttering under his breath, and so restless that restraint was necessary. His jaws were firmly locked, but with effort could be opened slightly. The neck was stiff, but there was no opisthotonos; knee-jerks increased on both sides, slight Babinski on the left side. He was given 27,000 units of antitoxin (diluted in saline solution) intravenously, and following this he had a severe chill and his temperature rose to 40° C. Immediately thereafter it fell to 37° and never subsequently rose above 37.8°. He was given chloral hydrate, 2 gm., and sodium bromide, 4 gm., every three hours by rectum, as he could not swallow. About 6.30 P.M. the patient began to clear up mentally. His neck continued very stiff. At 7.30 P.M. he was given two drops of oleum tiglii (croton oil). Voided 830 c.c. of urine during the day.

November 28, 1916. Patient improving; knee-jerks increased; slight Babinski, both sides. Head cannot be flexed beyond the plane of the body, the neck being quite rigid. Some headache. Bowels opened twice. Voided 800 c.c. of urine Chloral and bromide as yesterday.

November 29, 1916. Lies on his side curled up and sleeps most of the time. Takes nourishment well. Some headache. Neck is slightly stiff still. Chloral and bromides given only in one-third former dose, and by mouth.

December 1, 1916. Urticarial rash over the whole body. Itching relieved by carbolized vaseline.

December 2, 1916. Urticarial rash more pronounced. Relieved by 5 drops of pituitrin. Nauseated, but relieved by vomiting.

December 13, 1916. Convalescence has been uneventful. Went home today.

CASES OF TETANUS UNDER THE WRITER'S CARE.

No.	Date of admission.	Age and Sex.	Site of wound.	Date and nature of injury.	Incubation (time from injury to symptoms).	Time from symptoms to institution of efficient treatment.	Type of case.	Summary of treatment.		Result.
								Original injury.	Tetanus.	
1	Sept. 28, 1908	M 34	Abdomen and thigh	Sept. 24, 1908: Brush burns, hematoma; thrown from wagon and dragged	14 days	1 hour	Slow in onset, but severe convulsions	Ordinary antiseptic	Sedatives; antitoxin, subcutaneously (129,000 units) and intraspinally (3000 units); total antitoxin 132,000 units in 10 days	Recovered Oct. 31, 1908.
2	Sept. 23, 1908	M 55	Cranium	Sept. 23, 1908: Gunshot fracture temporal region and rupture of eye-ball	4 weeks	12 hours	Mild; slow in onset; no convulsions	Aseptic; no operation	Sedatives; antitoxin subcutaneously (31,500 units) in 8 days	Recovered; eye-ball enucleated Nov. 17, 1908.
3	Nov. 12, 1908	M 49	Foot	Nov. 4, 1908: Punctured wound by rusty nail	8 days	24 hours	Medium severity; no convulsions	Home treatment by ham fat, etc.	Sedatives; antitoxin intraspinally (3000 units) and later subcutaneously (27,000 units); total amount 30,000 units in six days	Recovered Nov. 18, 1908.
4	Dec. 19, 1908	M 19	Foot	Nov. 30, 1908: Punctured wound by splinters from mill floor	18 days	24 hours	Severe convulsions before admission	Wound dressed twice but patient never returned for further dressings	Three splinters removed from wound; sedatives; antitoxin intraspinally 3000, intraneurally 750 units, and into muscles around the wound 750 units; total in two days 4500 units	Recovered Dec. 31, 1908.
5	Oct. 16, 1909	M 63	Fingers	Sept. 28, 1909: Lacerations by machinery	18 days	24 hours	Severe; no convulsions	Ordinary antiseptic dressings	Fingers amputated; sedatives (chloretone); antitoxin intraspinally 1500 units	Died Oct. 19, 1909, after being comatose 24 hours.
6	Nov. 20, 1909	F 11	Knee	Nov. 11, 1909: Abrasion from fall	9 days	40 hours	Slow in onset; severe; convulsions late	Bread poultice at home	Sedatives (chloretone); antitoxin intraneurally 3 times (total 9000 units), intraspinally (1500 units) subcutaneously (3500 units); total antitoxin 13,500 units in 3 days	Died Nov. 22, 1909, after being comatose three hours.
7	Feb. 4, 1910	F 45	Toes	Jan. 23, 1910: Laceration; fall down stairs in bare feet	12 days	Few hours	Mild; slow onset; no convulsions	Neglected for ten days; then iodine	Sedatives; antitoxin, subcutaneously, 6000 units in three days	Recovered Feb. 19, 1910.
8	Feb. 21, 1910	F 23	Scalp	Jan. 26, 1910: Lacerated wound; hit by brick	19 days	1 week	Slow in onset; severe; no convulsions	Sutured; no drain	Abscess opened; sedatives; antitoxin intraspinally 3000 units, subcutaneously 21,500 units; total antitoxin 24,500 units in five days.	Recovered Mar. 8, 1910.

9	Oct. 3, 1910	F 6	Arm	Sept. 7, 1910: Vaccination	Unknown less than 24 days	3 days	Slow onset; very severe; 2 convulsions every hour	Treated at home by mother	Sedatives; antitoxin intraspinally 10,000 units (Oct. 5), subcutaneously 45,000 units; total antitoxin 55,000 units in two days	Died Oct. 5, 1910, in convulsions.
10	Feb. 4, 1911	M 6	Hand	Dec. 23, 1910: Laceration; fall on icy street	9 days	4 days	Severe convulsions	Sutured	Sedatives and magn. sulph. intraspinally; antitoxin intraspinally 9500 units; subcutaneously 10,000 units; total 19,500 units in two days	Died Feb. 7, 1911; in perfect relaxation.
11	Nov. 20, 1913	M 23	Sole of right foot	Nov. 12, 1913: Puncture by rusty nail	7 days	9 days	Trismus first sign; convulsion after admission	Untreated	Sedatives; antitoxin subcutaneously 18,500 in first 24 hours, intraspinally 10,000 second day, 9000 fifth day; intravenously 18,000 second day, 5000 fifth day	Died Nov. 25, 1913.
12	Oct. 29, 1915	F 17	Finger	Oct. 15, 1915: Puncture by machine needle in woolen mill	13 days	24 hours	Slow onset; local pains for five days, stiff neck for 1 day before admission	After twenty-four four hours opened; no antitoxin	Healed wound opened and cleaned; sedatives, large dose; antitoxin; intraspinally 10,000 units, intravenously 18,000 units, intramuscularly 6000 units	Recovered Nov. 10, 1915.
13*	Dec. 17, 1912	M 27	Right finger	Before Dec. 1, 1912: Crushed	9 days	9 days	Subacute onset; trismus epigastric pain, stiff legs; spasms 3 daysbeforetreatment	Bandaged by patient	Sedatives, very large doses; antitoxin intraspinally 8000 units, intravenously 28,500 units	Recovered Dec. 21, 1912; died Dec. 22, 1912, of pneumonia.
14*	April 11, 1914	M 58	Nose	April 7, 1914: Struck by piece of bone in fertilizing plant	3 days	1 day	Severe; facial paralysis	Abscess formed and this was opened fourth day	Sedatives; antitoxin intraspinally 18,000 units in two days; intravenously 13,500 in two days; into facial nerve, 1500 on second day	Died April 13, 1915, in coma.
15*	Nov. 17, 1914	M 18	Hand	Three weeks ago: scratch by wire nail	Over two weeks	5 days	Slow onset, with stiffness and pains in legs, trismus, spasms, opisthotonos and risus sardonicus on admission	Untreated	Sedatives, large doses; antitoxin intraspinally 6000; intravenously 15.000; subcutaneously 3000 units	Recovered Dec. 15, 1914.
16*	Oct. 20, 1915	F 34	Right knee	Oct. 4, 1915: Chafed in riding horseback	12 days	4 days	Subacute onset; pains in side, back muscles and neck; opisthotonos before treatment and constant spasms	Ointment applied by patient	Sedatives, very large doses; antitoxin intraspinally 9000 units, subcutaneously 20,000 units in six days.	Recovered Oct. 30, 1915.
17	Nov. 26, 1916	M 32	Foot	Nov. 16, 1916: Puncture by rusty nail	8 days	2 days	Slow onset with stiffness of cheeks, jaw and neck	Untreated	Sedatives; intraspinally 10,000 units, intravenously 27,000 units in first eighteen hours after admission	Recovered Dec. 13, 1916.
18	Sept. 18, 1920	M 13	Feet?	?	?	5 days	Slow onset, clonic spasms on admission and opisthotonos	Untreated	Sedatives; intraspinally 10,000 units, intravenously 35,000 units in first twelve hours after admission.	Recovered Oct. 1, 1920.

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* Cases 13, 14, 15, 16, seen in consultation; all the others treated in the Episcopal Hospital, Philadelphia.

NITROUS OXIDE AND OXYGEN WITH REBREATHING IN SURGERY

By MAURICE C. BOND, D.D.S.
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The anesthesia produced by the combination of nitrous oxide and oxygen with rebreathing, supplemented by ether when indicated, ranks above ether so far as safety to life is concerned. The induction period under nitrous oxide is much shorter and easier than with ether. In point of after-effects, it takes precedence over all other agents providing it is administered with a fair degree of care. Premedication is advisable as the cases run more smoothly, necessitating a decrease in the amount of ether needed and resulting in a more nearly ideal recovery providing the patient is given an average dose of morphin (gr. $\frac{1}{4}$) and atropin (gr. $\frac{1}{150}$) one-half hour or chloretone (gr. 15) one hour before the beginning of the anesthesia.

Notwithstanding the fact that nitrous oxide and oxygen is the "ideal" anesthetic, there are cases where this anesthetic is contra-indicated. Children are not favorable subjects for this form of anesthetic because of their miniature musculature which makes breathing into the bag difficult. In some abdominal operations in which complete relaxation is required one should resort to the use of some other anesthetic. As yet a perfected technic has not been developed for tonsillectomies. Athletes and persons addicted to the use of alcoholic stimulants take nitrous oxide and oxygen very poorly. Ether is indicated in these cases.

Satisfactory results with nitrous oxid and oxygen depend largely on the correct technic of administration and here it might be well to add that the anesthetist should be given ample time to bring the patient to the surgical stage of anesthesia. The patient is rendered unconscious in approximately two minutes, yet it is nearly ten minutes before the body is sufficiently saturated with the nitrous oxide to permit the beginning of an abdominal operation. If the surgeon then feels that the patient is not sufficiently relaxed, a small amount of ether is added to the nitrous oxid and oxygen combination. In beginning the administration, the bag is three-quarters filled with nitrous oxide and the patient allowed to breath this gas for about two minutes. Nitrous oxide alone is administered in the initial stage because it lessens the induction period. Too much stress cannot be laid on the correct adaptation of the face-piece to the case at hand. If air is allowed to get in at the sides, the anesthesia will be uneven and may be followed by a period of excitement. Following the administration of the pure nitrous oxide sufficient oxygen is added to prevent cyanosis. The valve on the face-piece is closed after the first few inspirations thereby instituting rebreathing. From time to time this valve is opened in order to expel the gas the patient has been breathing, thereby allowing for a fresh mixture. The anesthetist has three indicators by which to determine the correct percentage of gases suitable to the case: namely the color index, the bag containing the mixture of gases and the mixing chamber when the Gwathmey machine is used. The gases bubble through a glass mixing chamber which contains water. The fulness of the bag indicates whether the patient should be receiving the amount of gas coming through four of the nitrous oxide holes and one hole of oxygen or whether five holes of nitrous oxide and two of oxygen is the necessary amount. As the cases progress the percentage remains the same with few exceptions, but the volume of the gases can be reduced to four holes of nitrous oxide and one of oxygen.

The percentage of complications following nitrous oxide and oxygen is very small. This is one of the striking points of the method. The patient is conscious within a few minutes following the removal of the face-piece and the majority of cases are not sick. Dr. Ashhurst's records show that of one hundred and one cases, eleven vomited, or approximately eleven per cent., while following the administration of ether, two hundred and sixty-two out of four hundred and sixty-one cases vomited, or fifty-three per cent. The character of the operations performed under nitrous oxide and oxygen varied, such as reduction of fractures; incision and drainage of abscesses of the hand, forearm, ilium, pelvis, appendix and subpectoral regions; appendectomies; suprapubic prostatectomies, herniotomies, intussusception of the ileum; leg and arm amputations; carcinoma of vagina; perforated duodenal ulcers, etc. Occasionally the patient complains of headache, which is probably due to too much rebreathing. There is no appreciable rise in temperature following the operation such as is so common after ether. Gwathmey believes that nitrous oxide and oxygen exert no unfavorable effect upon the urinary organs.

I believe the time is not far distant when surgeons will prefer this form of anesthetic for the majority of operative procedures. The technic as well as the machines are gradually being improved, and when the price of the anesthetic is so modified that long operations can be done at a reasonable cost nitrous oxide and oxygen anesthesia should be much more generally employed. At the present writing both oxygen and nitrous oxide cost three cents per gallon, making the cost of administration per hour three dollars. I recall a case where I administered this anesthetic for three hours and forty minutes. The nature of the operation was intussusception of the ileum.[1] The results were very encouraging and I trust that in the future a larger per cent. of abdominal operations, especially in septic patients, will be done under nitrous oxide and oxygen.

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[1] See p. 454 of this volume.

THE TREATMENT OF SHOCK AND HEMORRHAGE

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THE purpose of this article is to deal with shock and hemorrhage as the result of wounds and to set forth a few personal observations made, during a course of study, at the Central Medical Department Laboratories at Dijon, France, under the tutelage of Major W. B. Cannon, M.R.C., U. S. Army, and during my work, while on Emergency Medical Team No. 130, at Evacuation Hospital No. 8.

In order properly and logically to discuss the treatment of shock, it is somewhat necessary briefly to discuss some of the causes and some of the most important of the phenomena present.

However, it is not my purpose to mention the many well-known theoretical causes of shock, and I intend simply to enumerate the most important predisposing factors which seem to have a definite relation to the treatment.

Of these, it may be said that atmospheric conditions constitute one of the most important. Loss of bodily heat is a main factor in shock, and any circumstances which augment this loss of heat, are of vital importance for consideration. Hence, a low atmospheric temperature predisposes to shock and augments it, if shock is already present, by inducing and augmenting circulatory stasis, especially in the capillaries. Also, an atmosphere of a very high humidity is very apt to promote the activity of the sweat glands, thereby causing a loss of body heat through perspiration. Therefore, the longer the exposure to such atmospheric conditions the greater the shock will be.

The general physical condition and resistance are other important factors. For instance, a man who has not had any sleep for several days, who has had food but irregularly and in questionable amounts for several days, and who has had but little water, will be far more susceptible to shock than a man who has not experienced the above-mentioned hardships.

Finally, wounds themselves must be considered in regard to their number, location and character. Other things being equal a single wound produces less shock than multiple wounds. Clean-cut wounds with but slight tissue destruction, as, for instance, a perforating wound of the ankle, due to a machine-gun bullet, cause but little or no shock. However, a wound with marked tissue destruction, in which there is tearing, bruising and mutilation of tissues, causes profound shock which often proves fatal. Such a wound is that made by an irregular fragment of a large shell, in which there is tearing and crushing of the muscles of the thigh with, perhaps, a compound fracture of the femur.

As to the symptoms of shock, I do not intend to enumerate them here, for they are only too well known. However, I do wish to mention some of the important phenomena which are present in the shock state and which are of importance from the standpoint of treatment. In this respect the circulatory phenomena attract our special attention.

The main index as to the degree of shock is the blood-pressure and it will always be found that the greater the degree of shock the lower the arterial pressure. It is well known that blood-pressure is due to three things, namely, the force exerted upon the blood by the heart, peripheral resistance to the flow of blood and the volume of blood. An injury in a perfectly healthy individual does not, in any way, alter the cardiac force nor the peripheral resistance and it must therefore be considered that the volume is at fault. In other words there is a loss of volume of the circulating blood and this lost blood is to be found in the capillaries. By making a capillary and a venous blood count in shock it will be found that the capillary count will be, by far, the higher. Therefore, we have a concentration of blood in the capillaries or a capillary stasis, in which the blood does not get back to the heart in sufficient quantities to keep up the arterial pressure. This condition is practically a "potential hemorrhage."

It is the capillary system that supplies blood to the tissues, and the rest of the circulatory apparatus simply keeps up the flow of blood for the capillaries. The higher the arterial pressure the better the capillary circulation. However, we have already shown that in shock we have a low blood-pressure and a stagnation of blood in the capillaries. Therefore the capillaries are unable properly to supply the tissues with blood and we have inactive tissues due to oxygen starvation. This results in the tissues producing non-volatile acids, which fail to be oxidized because of a diminished oxygen supply to the tissues, and which enter the blood and break up the sodium bicarbonate of the blood by combining with the sodium and driving off carbon dioxide. For example, lactic acid is formed and thrown into the blood-stream where it breaks up the sodium bicarbonate to form sodium lactate and carbon dioxide. An examination of the blood therefore reveals a diminished sodium bicarbonate content and we have a reduction of the alkaline reserve of the blood, which, when it gets below its normal limits, causes acidosis. However, there is also another factor to be considered, namely, the acid substances which are given off from masses of injured muscles. These acid substances are also absorbed into the circulation and help to produce acidosis. In fact, it is claimed that toxic substances given off from the injured tissues are largely responsible for the shock state and this has been called traumatic toxemia.

Now, there is a definite relation between the acidosis and the degree of low blood-pressure. It has been found that a systolic blood-pressure above 80 mm. of mercury is sufficient to give an adequate supply of oxygen to the tissues and hence does not produce acidosis. However, a systolic blood-pressure below 80 mm. of mercury produces an insufficient oxygen supply to the tissues and acidosis results. Therefore a systolic pressure of 80 mm. of mercury is called the critical pressure. In regard to this, the time element is very important for if a pressure below the critical level is allowed to persist we very quickly get permanent damage to the tissues, and especially to the vital centers in the brain. Hence the importance of immediate interference to maintain the pressure above the critical level.

And now to briefly summarize these changes, we may say that we have an injury, an absorption of toxic substances from the injured area or a traumatic toxemia, which causes a low blood-pressure and a circulatory stasis with the resulting oxygen starvation of the tissues and an acidosis, which in turn, if allowed to persist, causes permanent damage to all the tissues of the body.

What has been said in regard to shock applies likewise to hemorrhage, for the phenomena are practically the same in both instances. Most cases of shock from wounds are associated, to a greater or less degree, with hemorrhage. Shock associated with severe hemorrhage will naturally be more grave, for the loss of the oxygen-carrying constituents of the blood will, of course, augment the oxygen starvation already induced by the shock. For this reason the critical level of the blood-pressure, in shock associated with hemorrhage, is higher, having been estimated at 90 mm. of mercury.

Now we come to the treatment of this condition and this is covered by but three words, namely heat, rest and fluids.

As to heat we should try to prevent any further loss of bodily heat and try to make up for that already lost. We can, in a measure, accomplish the former by preventing sweating, keeping the skin and clothing dry and by avoiding undue exposure in the examination of patients. In order to make up for the bodily heat already lost we must apply external heat.

In this respect one must be cautioned not to overheat patients as this often causes profuse sweating with a subsequent loss of heat, thereby defeating our purpose. The methods employed with the most success at Evacuation Hospital No. 8 were as follows: First, hot drinks were given to all shocked patients, providing there were no abdominal wounds nor any nausea and vomiting. Second, hot water bottles were placed about the patient in such a way that all surfaces of the hot water bottles were utilized. Third, small alcohol stoves were used. These were placed under the beds with iron mattresses and the blankets were then draped so that they touched the floor on all sides of the bed, thereby preventing escape of the warm air.

Following upon or simultaneous with the application of external heat an attempt was made to keep the patient at complete rest in order to prevent sweating with loss of bodily heat and to prevent tissues that were already starved for oxygen, from demanding an extra supply of oxygen. As most of the patients were extremely restless and tossed around a great deal it was deemed advisable to administer morphine sulphate to its full physiologic effect.

The importance of treatment by forced fluids cannot be overestimated for it is well known that, following a hemorrhage, the fluids in the tissues outside of the circulation are poured into the circulation to make up for the lost volume due to the hemorrhage. In other words there is a natural recovery. If the hemorrhage is but slight this fluid in the tissues will, in many cases, be sufficient to make up for the lost volume, but if the hemorrhage is very severe we will need outside assistance and more especially if the fluid reserve of the tissues is low to start with, as it was in most of the cases that I saw. Therefore treatment by means of forced fluids proves efficacious by increasing the volume of fluid in the vessels and by increasing the circulation and thus the corpuscles, though reduced in numbers, are put to greater use through a more rapid circulation.

Fluids were administered in two ways, first by the digestive system and under the skin and secondly by introducing the fluid into the veins. All patients, who did not have abdominal wounds and who were free from nausea, were given forced fluids by mouth. Many, however, became nauseated and it was necessary, in these cases, to give enteroclysis or hypodermoclysis of salt solution. As to the introduction of fluid into the veins it was found that any intravenous treatment proved harmful if given before the patient had begun to recuperate to some degree under the influence of heat and rest. This method was also harmful unless it was first determined that an increase in blood-pressure would not induce hemorrhage or augment it if already present. When immediate operation was imperative, intravenous treatment proved most efficacious if administered just before or at the start of the operation and oftentimes it was necessary to repeat it afterward. In fact, a frequent repetition of this form of treatment should be resorted to if necessary and so long as recovery is at all possible.

The fluids used in this therapy were normal salt solution, gum salt solution and blood, the idea being to have a solution readily accessible and which would stay in the vessels the longest period of time and produce the most permanent rise in arterial pressure.

As to the relative values of the methods employed, it is without doubt that the transfusion of blood was, by far, the best. Most of the cases of shock were associated to a more or less degree with hemorrhage and in these cases we were dealing not only with a loss in volume of circulating fluid but also a loss of corpuscles. Hence the blood not only supplied volume but likewise oxygen-carrying constituents to the circulation. Besides, blood is the normal content of the vessels and will remain in the vessels and thus produce a permanent rise in the blood-pressure. Also the simplicity of the method of transfusion employed, the sodium citrate method, made this the treatment of choice whenever donors were available.

I consider fluids by the digestive system and under the skin second to transfusion because these methods can be employed in conjunction with any other treatment desired and because I found that salt solution, administered in this way, caused a more permanent rise of blood-pressure than salt solution into the veins, possibly because it is taken from the tissues into the vessels more slowly as it is needed, and possibly because it undergoes some changes in the tissues whereby it is more suited to remain in the blood stream a longer time.

Normal saline infusions are considered next because they were but temporary measures and it was found that an infusion of from 500 to 1000 c.c. of normal saline solution produced but a temporary rise in blood-pressure due to the fact that the solution would not remain in the vessels any length of time.

Gum acacia solution or gum salt solution, as it is called, is considered last because it not only proved useless but actually proved harmful and even fatal in many cases, when introduced into the veins. I have heard no good logical reasons expressed for this with the exception that some say the cases, in which it proved fatal, had been in shock from twenty-four to forty-eight hours and the tissues had been in a state of oxygen starvation too long for gum salt or anything else to do any good. Others claim that it proved fatal because it was used in cases with a concealed gas bacillus infection. However, my cases were not selected but taken as they were admitted to the shock ward and, while some of them may have been in a state of oxygen starvation too long or may have had concealed gas bacillus infections, I am sure that the vast majority of them were cases of recent true shock. Likewise the gum saline solution was neutralized and properly prepared and was given very slowly and carefully. Yet so many fatalities occurred that its use was soon given up for blood when available or salt solution. Certainly gum salt lowered blood-pressure in most cases instead of raising it and those cases in which the arterial pressure was increased by it, showed but temporary improvement and the pressure speedily dropped back to its former level or, more often, far below it.

Here I wish to interpose but a word concerning what may be termed the surgical treatment of shock. Some cases show no tendency whatever to improvement under medical treatment and either lose ground or remain in a stationary condition. The question here arises as to what length of time we should continue medical treatment if no improvement is shown and, in such cases, when should surgical treatment be instituted. The length of time for medical treatment will vary in different cases and no definite time can be set forth. However, if after several hours no improvement is noted and all medical resources are exhausted, one is justified in operating even though the patient is a very poor surgical risk, for it is a case of either allowing him to die without interference or doing something radical as a last chance. Hence, in such cases it may be possible to overcome the shock and save the patient's life by surgical removal of the source of the toxins.

Now in conclusion, it may be said that first, heat and rest should be employed in all cases of shock and hemorrhage and will be found to be sufficient treatment in a large percentage of cases of true shock. Second, cases with a systolic blood-pressure of 80 mm. of mercury or above, which show a tendency to improvement in one hour under the influence of rest and warmth, as evidenced by a permanent rise in the systolic pressure, will be benefited by saline infusions and salt solution by the digestive system. Third, cases with a systolic pressure of 80 mm. of mercury or below, which fail to show improvement in one hour under the influence of rest and warmth, as evidenced by the blood-pressure, will not be benefited by infusions but should be given blood transfusions. Finally, all extreme cases, which are apparently in a dying condition, should be given the only chance they have of recovery, namely, a blood transfusion and in many instances a veritably lifeless man will rally and be truly born anew.

THE METHOD OF RECORDING SURGICAL OPERATIONS AT THE FRONT[1]

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"They do these things better in France," is a phrase which had passed into history many years before the great German War, but the truth of which has been verified anew in many respects during the experiences of the last four years. Especially, from the military point of view, did I find this to be the case with the methods of recording surgical operations during the hurry of work in advanced posts. Surgical Team No. 23, U. S. Army, of which I was the senior member, with Lieut. Henry S. Kerchner as my assistant, together with two nurses, an anesthetist, and two enlisted men as operating room assistants, worked for three months in a French "Autochir," and it became our duty to put on record in French an account of each operation as it was done. So explicit is the French language, and so nicely does it lend itself to terse and accurate expression, that when later our activities were transferred to an Evacuation Hospital of the United States Army we carried along with us certain habits of expression which might well be officially adopted into the military code of armies other than the French.

In order for any statements, spoken or written, to be clear and intelligible, it is first and foremost necessary for the speaker or writer to know definitely in his own mind the points he wishes to have recorded and the order of their statement, and for him to employ definite and fixed terms of expression. These habits the French surgeons have acquired, and they gladly pointed out to us the important facts which should be recorded, as well as the value and significance of certain habitual abbreviations.

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[1] Read before the Philadelphia Academy of Surgery, May 5, 1919. Reprinted from the Annals of Surgery, 1919, vol. ii, page 241.

Nom	Prénoms	Grade	Corps, Compagnie, etc.

Diagnostic sommaire

Ci-dessous, souligner au crayon de couleur les mentions utiles

Blessé le....................

Injections antitétan.
  1re faite le....
  1re à faire....
  2e faite le....
  2e à faire....

Transportable
  assis
  couchè
  à courte distance
  à zone des étapes
  à interieur

Catég. speciales
  Officier
  Allié
  Musulman
  Indigène
  Ennemi

(Opération importante)

......faite le.....

Soins à donner en cours de route..

Règime..........

à sonder

Pansement
  faite le..
  à faire le.

Services Destinataires
  A. Chirurgie générale: *grands blessés ou soins op.*
  B. Chirurgie générale: *petits blessés ou soins non op.*
  C. Chir. spéc. *Maxillo-faciale*
  D. Chir. spéc. *Urinaire*
  E. Chir. spéc. *Orthopédie*
  F. Chir. spéc. *Syst. nerveux*
  G. Chir. spéc. *Ophthalmologie*
  H. Chir. spéc. *Oto. rhino-lar.*
  I. *Prothèse dentaire*
  K. *Physio* ou *Mécanotherapie*
  L. Médecine générale
  M. Méd. spéc. *contagieux*
  N. Méd. spéc. *Vénériens*
  O. Méd. spéc. *Aff. cut.*
  P. Méd. spéc. *Psychiat.*
  R. Méd. spéc. *Aff. nerv.*
  S. *Hôpital sanitaire*
  T. *Convalescents.*
  Z. *Eclopés* ou petits malades (zones des étapes)

FIG. 3.—The "Pochette" or French Field Envelope.

The patients would arrive at the operating room of the autochir with their "pochette" (the diagnosis envelope) already made out. This had been done at the "Triage" (sorting center) of the autochir, the patient's name, rank, etc., and the "diagnostic sommaire" (summary diagnosis) being recorded on the outside of the envelope (Fig. 1). Here also was recorded the *date and hour of injury*, whether or not the *injection of antitetanic serum* had been given; and when the patient was dismissed from the operating room or later when discharged from the ward, other notable facts were added on the outside of the envelope, such as the *nature and date of any important operation;* how the patient was to be *transported* (sitting or recumbent); whether he was to be transported only a short distance, or to the intermediate zone, or to the interior; whether he was an officer, a member of one of the allied armies, a Musselman (important because no wine could be taken by such patients), etc.; as well as *treatment required during transport* (diet, *i. e.*, regular, special, or liquid); whether or not *catheterization* was required; the *date of the last dressing* of the wound, and the date when it should be dressed next; and finally the special service to which the patient was to be assigned on arrival at the rear. Most of these facts could be indicated by underscoring or striking out with a colored pencil.

Inside the pochette, on arrival at the operating room, was a slip of paper on which the radiologist had recorded the results of his fluoroscopic examinations. These radioscopic diagnoses were models of accuracy and neatness, considering the great speed with which the examination had to be made.

For the space of one minute or thereabout, intervening between the time the surgeon could turn his attention from the last patient to the patient next in order, opportunity was afforded to scan the summary diagnosis on the outside of the envelope, and to read the radiologist's diagnosis. Toward the conclusion of each operation the surgeon would then dictate in French (or, if an American surgeon, then in patois), what was to be recorded for the information of the surgeon into whose care the patient should come at his next station. This dictation, taken down by a clerk (or in emergency written by the surgeon himself, or by any of his staff who understood French), was written on another scrap of paper (paper was valuable and scarce); this also was placed in the *pochette*, and accompanied the patient to his permanent hospital, where "fair copies" could be made of all the scraps of paper which had accumulated on the journey. When time permitted, this fair copy was made in the office of the autochir, in manuscript, on the "Billet d'Hôpital à Coupons" (a folded form on which the Field Medical Card of the United States Army is modelled), and this also was placed in the *pochette*, and was signed by the "Médecin Traitant" or Ward Surgeon.

The French surgeons made a habit of keeping for their private records a copy of their dictation, and needless to say this habit also was gladly adopted by the American teams working with the French. A small notebook, carried in the pocket, furnished all the scraps of paper required; alternate pages were written on, and the intervening carbon copies were torn out and placed in the *pochette.* It is true that the Médecin-Chef had occasion once to complain that the carbon copies placed in the pochette were "presque illegibles," but it is believed this applied to copies furnished by one of the French teams, at a time when the supply of carbon paper had been nearly exhausted.

The points to be recorded on these scraps of paper were the following, and in the following order, as near as might be: The formation where the operation was done, the patient's name, rank, etc., the date and hour of injury, the date and hour of operation, the summary diagnosis, including the nature of the missile; the *x*-ray report (this was always repeated in condensed form under the description of the operation, in addition to the original report furnished by the radiologist); a concise account of the operation and the condition of the wounded parts; the dressing of the wound; whether the patient was to be evacuated at once or to be held for further observation; and the means of his evacuation (*i. e.*, by hospital train, by motor ambulance to the nearest hospital center; or, if a trivial injury, to the center near the front where such patients were sent for recuperation before immediate return to the lines); and, at the end, the operator's name.

The autochir with which we were longest associated (Ambulance Chirurgicale Automobile No. 6, of which the Médecin-Chef was M. le Professeur Agrégé J. Guyot, of the University of Bordeaux) was situated in the outskirts of a village called Crèvecoeur-le-Grand, in Picardy, in back of Montdidier (and Cantigny), south of Amiens, and about 20 kilometers north of Beauvais, the nearest large town, which before the war had a population of about 20,000. The slightly wounded ("minima") were evacuated usually to Songeons, a town toward the west, in the intermediate zone ("Zone des Étapes"); but if the railroad happened to be cut by the German bombing expeditions, they were sent to Grandvilliers, nearer at hand; the severely wounded, including almost all the fractures, were sent by ambulance to Beauvais, which was a large hospital center, and a flourishing one until the German bombing expeditions made life there, even in hospitals, rather precarious; while those patients who could travel to the interior of France were taken by ambulance to the H. O. E.[1] (Evacuation Hospital) about a kilo and a half distant on the Froissy road, and were there loaded on the Train Sanitaire.

Appended are a few typical records in Americanized French, from our service at Autochir 6, and some corresponding records, in Gallicized English from our service at Evacuation Hospital 6, U. S. A. (Souilly, south of Verdun). Explanations of the abbreviations are given in tabular form, for convenience of reference.

The United States Army did well to copy their Field Envelope and their Field Medical Card from the French; but in one essential point the American surgeons were inferior to the French in training: they did not know, and they had not been taught the important points which should be recorded. And the Field Medical Card was faulty in that it did not contain any place for recording the *hour of injury*. Space was provided for recording the hour *first aid* was given, but it is of no use to know the time of this unless it is known how long a period elapsed between injury and first aid. Moreover, experience has shown that "first aid" was very much less important as a factor in preventing infection of the wound than the first operation—excision, débridement, and extraction; and in the future it is the hour of injury, rather than that of first aid, which should be carefully recorded. The patient almost always knows approximately the hour of his injury, even though he may not know the day of the week or month, and these latter can be supplied by the surgeon. The American Field Medical Card is also troublesome because too long to fit snugly in the Field Envelope; and, being made of cardboard, it is impossible for the surgeon to take a carbon copy from it, if he makes his record directly upon it, as was evidently the intention, and as was the practice as long as the supply of Field Medical Cards lasted.

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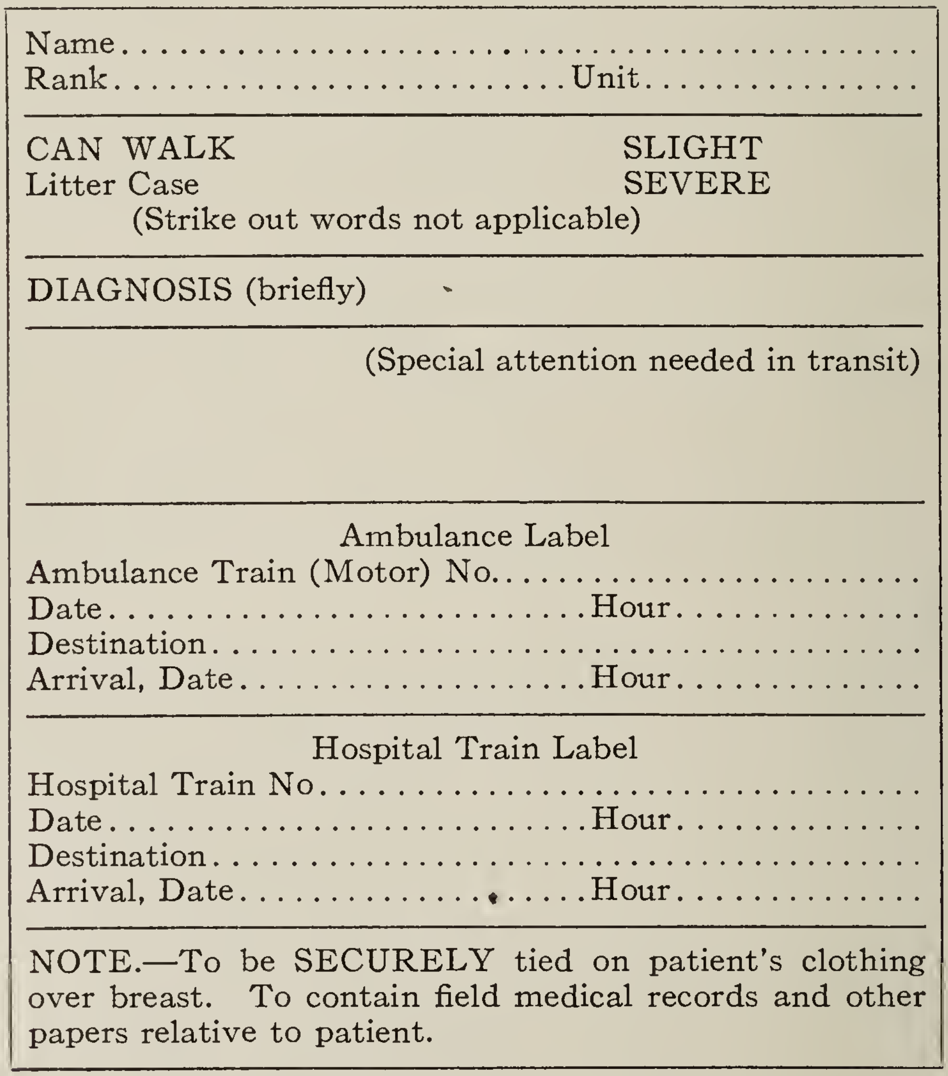
[1] The formula H. O. E. is derived from the term Hôpital d'Evacuation, which in the early days of the war was abbreviated as Ho. E., and by the corruption of sign painters subsequently degenerated into H. O. E. At least this is the explanation given by the French themselves.



Name..

Rank.................................Unit......................

CAN WALK SLIGHT

Litter Case SEVERE

(Strike out words not applicable)

DIAGNOSIS (briefly)

(Special attention needed in transit)

Ambulance Label

Ambulance Train (Motor) No..................................

Date.................................Hour....................

Destination..

Arrival, Date..........................Hour....................

Hospital Train Label

Hospital Train No...

Date.................................Hour....................

Destination..

Arrival, Date..........................Hour....................

NOTE.—To be SECURELY tied on patient's clothing over breast. To contain field medical records and other papers relative to patient.

FIG. 4.—Face of U. S. Field Envelope.

The habit of the American army in recording all war wounds as "gunshot wounds" (G. S. W.), is another stumbling block to an accurate and terse statement of facts, unless it be considered of extreme importance (which it is not) to distinguish between these and the almost unknown bayonet or sabre wounds. The important point for record is the *nature of the missile*, and especially whether it was a shell fragment or a ball; the difference between a wound by a rifle ball and a machine gun bullet is negligible, except for medico-legal investigations, but the difference between a wound by a shell fragment and any kind of a bullet may mean the difference between gas gangrene and rapid healing, and this fact (nature of the missile) should always be recorded. Likewise the difference between wounds by shell fragments, grenade fragments, and fragments from aëroplane bombs, is neglible, except in a sentimental or historical sense. The habit of marking all these wounds merely as g. s. w. makes the surgeon in too many cases feel he is absolved from any further record. It is more satisfactory in every way, for the clinical history

APPROVED ABBREVIATIONS

Morphia	M.
Anti-Tetanic Serum	A.T.S.
Gunshot Wound	G.S.W.
Fracture, Simple	F.S.
Fracture, Compound	F.C.
Fracture, Compound, Comminuted	F.C.C.
Not yet diagnosed or Undetermined	N.Y.D.
Fever of Unknown Origin	F.U.O.
Disordered Action of Heart	D.A.H.
Slight	O.
Severe	S.

FIELD MEDICAL CARD

Name..........................
(Block letters)
Rank...............No...........
Regiment or Staff Corps......Co...

Sick Wounded
Line of Duty—Yes or No
(Strike out descriptions which do not apply)

Field Hosp. No... Evac. Hosp. No...
Dress. Station No...Camp Hosp. No.
Date of Admission................
Morphia
Dose and Time
A. T. Serum
Dose and Date
Diagnosis

Base Hospital No..................
Diagnosis (if altered from above)

Base Hospital No.................
Diagnosis (if altered from above)

FIG. 5.—Outside of U. S. Army "Field Medical Card" (adopted from the French "Billet d'Hôpital à Coupons") to be folded in three and carried in Field Envelope. The column headed "Field Medical Card" must be the center third when the card is folded. The column "Approved Abbreviations" on the left and a blank column on the right form the remaining two-thirds.

of the case, if the injury is recorded as "wound by ball" or "wound by shell fragment." It is worth while to note that wounds by "shrapnel" are quite rare, and that the term is carelessly and erroneously used as equivalent to shell fragment. It is also important to distinguish in the record between penetrating and perforating wounds: in the former the missile has lodged, in the latter it has passed all the way through. After this initial formula "Wound by shell fragment penetrating right thigh" or "Wound by ball perforating left forearm," should appear, in the summary diagnosis, the main complication, as "with fracture of femur, middle third," or "with section of ulnar nerve and artery above wrist."

Date of entry and medical unit receiving patient must be recorded immediately on admission and signed by M.O.		Brief Clinical Notes
Field Evacuation } Hospital No.... Camp Date of Entry..........	Base Hospital No..... Date of Entry.......	Base Hospital No...... Date of Entry........

This card must accompany the patient, attached to his clothing. It must not be destroyed. It will be transmitted with the patient if he is evacuated to the U. S. Temperature charts or additional clinical notes may be sent with this card in the same envelope.

Fig. 6.—Reverse of U. S. Army Field Medical Card. To be folded in three, leaving three columns for clinical notes.

Next should come a statement of the positions of the wounds of entrance and of exit if the latter exist. If either wound is abnormally large and ragged it should be so stated. A concise and abbreviated description of the operation follows. In most cases it is sufficient to state "excision, débridement, extraction," as these terms denote definite and well-recognized procedures. If the foreign body is not found, this fact should be noted; or if it was so small as to be negligible and was not searched for, it should be so stated. If counterincision is made to facilitate extraction of the missile, or for the purposes of drainage, the fact should be noted. If arteries have been sectioned, the fact that they have been ligated should be a part of the history; or if a nerve has been injured its condition (contused, or sectioned) and the treatment adopted should be indicated. In cases of fracture the treatment of the fragments should be recorded, especially whether or not fragments have been removed, and the extent of the esquillectomy (large, medium, or parsimonious). The antiseptic employed also should be noted. It is sufficient to record it very summarily—as iodin, Dakin, Mencière. The method of drainage (tube or wick), the dressings and splints should form a part of the record.

Now to get all these facts in the record requires not only systematic dictation, but it requires a clerk with a reasonable knowledge of surgical terms, a good general education, and above all a good handwriting, and speed and accuracy. It is important to get through with as many operations hourly as is humanly possible. To demand this clerical work of the anesthetist or of one of the surgical team needlessly delays the commencement of the next operation. The French reckoned that the utmost speed conceivable would not permit the surgeons to average more than four patients in an hour, and they were quite well satisfied when a speed of three hourly was maintained. This requires not only that the patients be supplied to the operating room at that speed (which demands machine-like working of the triage, the preparation rooms, the sterilizing plant, and the fluoroscopic examinations), but it requires also that the patients leave the operating room at the same speed. While we were with the French it was possible at most times to maintain this average (three patients an hour, or about 24 patients in the eight-hour period of operating), with the aid of the clerks they supplied and the wonderful coördination maintained in the accessory services mentioned above. But with the American army we never could average more than two patients hourly except for very short periods at a time.

I would recommend, therefore, that before the next war the following modifications of the methods now in use of recording surgical operations at the front be adopted:

1. A field card of thin, tough, non-bibulous paper, of a size to fit easily inside the field envelope.

2. A space on the field card for recording the time of injury.

3. The records to be made in indelible pencil, and the surgeon encouraged to keep for his personal use a carbon copy of the records.

4. The abolition of the cumbersome term "gunshot wound" and the substitution for it of the term "wound by shell fragment (or ball, or grenade fragment, or shrapnel, or airplane bomb, etc.)."

5. The adoption of the French terms "Orifice of Entry" and "Orifice of Sortie," abbreviated as O. E. and O. S., as more terse than "Wound of Entrance" and "Wound of Exit" which cannot be so intelligibly abbreviated.

6. The provision of trained clerks for recording the surgeon's dictation. These clerks could be trained under the auspices of the Red Cross and would prove a valuable addition to the surgical teams for work in the advanced area.

Examples of Operations Recorded in French

A. C. A. 6.
Peruy, Charles, Cpl. 49 Inf., Cie. 3.
Bl. 8. 6. 18, 12 h.
Op. 8. 6. 18, ½ h.
Plaies multiples par E. O.
Radiog.: Bras g.: petit éclat superf., face ext.
Main g.: plusieurs tres petites éclats.
Cuisse g.: éclat face int. du femur niveau trochanter minor.
Region lombaire et pied g., neg.

1. Excis. et débrid. d'un plaie region lombaire g., hematome; projectile non trouvé; excision d'un bouillie des muscles lombaires. Mèches iode.
2. Débrid. O. E. niveau troch. major face post. Fracture découpée de la troch. minor. Extraction d'un projectile tranchant. Drain et mèches iode.
3. Débr. O. E. et S. region malleolaire post. g. Trajet a passé en avant du tendon d'Achille et il y a section de l'artere et veins tib. post. Ligatures. Nerf intact. Mèches de part en part, iode.
4. Débr. et extr. d'un projectile de la muscle triceps au dessus du condyle ext. de l'humerus g. Mèch iode.
5. Nettoyage de la main.

A Garder.

A. C. A. 6.
Hoffman, Carl, Soldat, 1e. Eng., U. S. Army.
Bl. 28. 4. 18, 12 h.
Op. 29. 4. 18, 17 h.
Plaie penetrante epaule gauche par E. O. O. E. 2.5 cm. en avant de l'acromion.

Radio: Petit projectile (grosse tête d'epingle) repère à 3 cm. de profondeur (bras le long du corps). Ce projectile se profile entre l'acromion et la tête humerale, au contact de la tête, probablement intra-articulaire.
Incision, extraction du projectile sous l'ecran, logé dans la capsule au dehors du tendon bicipitale. Suture des muscles et de la peau.
Evacuer, T. S. Crèvecoeur.

A. C. A. 6.
Naouar, Oub Abder Kader, 2e, Cl., 7e Tir.
Bl. 26, 4, 18.
Op. 27, 4, 18, 22 h.
Plaie perforante cuisse gauche par balle, entré en avant, sortie posterieure en dehors du femur.
Débridement, excision large. Drain de part en part. Mencière.
Plaie en seton region lombaire gauche.
Débr., excis., Mencière.
A Garder.

A. C. A. 6.
Hains, Theodor, 34e. Inf. Allemand.
Bl. 9. 6. 18, à nuit.
Op. 13. 6. 18, 14½ h.
Plaies penetrantes, crâne, épaule et bras g., par E. O. Les plaies de la tête et du creux axillaire g. sont infestées par des vers.
1. Plaie pen, pli post. de l'aiselle g. Exc., débr., extr. du projectile par contreouverture au pli anterieur de d'aiselle. Drain de part en part.
2. Seton du bras g. Excis. Mèche de part en part.
3. Grande plaie du cuir chevalu, region parietale gauche. Excision, extraction du projectile fixé dans le crâne. Autoplastie du cuir chevalu.
4. Éclatement oeil g. Enucleation et esq., fracture orbitale.
5. Plusieurs plaies superficielles de la face g., excision et sutures.
A Garder.

Examples of Operations Recorded in English

E. H. 6.
Switzer, Joseph, 61st Inf., Co. C.
Wd. 10. 12. 18, 6 h.
Op. 10. 12. 18, 19 h.
Wd. by ball entering outer face l. thigh, leaving inner face l. thigh, middle ⅓, traversing penis with rupture of corpus cavernosum and laceration of foreskin, then causing tangential wd. r. thigh.
X-ray neg.
Op. ether. Nett. of wds. Suture of penis. Iodin.
Evacuate.

E. H. 6.
Ricotte, Antonio, 61st Inf., Co. F.
Wd. 10. 16. 18, 18 h.
Op. 10. 17. 18, 17¼ h.
Wd. by S. F. perforating face and skull, with F. C. C. rt. mastoid region. Patient has been in shock ward since admission. Has cerebral symptoms, some rigidity of neck. Rt. eye marked exophthalmos. Punctured wd. to left of root of nose. Brain oozing from external auditory meatus. Missile probably entered at nose and made its exit through mastoid. *X*-ray neg.
Op. Ether. Excision of scalp wd., of prolapsed brain, and of all depressed and soiled fragments of bone. Complete closure of scalp by autoplasty. Iodin.
Hold.

E. H. 6.
Carden, Stanley, 316th Inf., Co. C.
Wd. 10. 11. 18, 13 h.
Op. 10. 13. 18, 2 h.
Wd. by S. F. penetrating rt. buttock.
X-ray, buttock: F. B. 20 x 15 mm., 70 mm. deep.
Op., ether. Debr. and extr. from beneath sciatic nerve. Persistent bleeding from branch of gluteal artery controlled with great difficulty by suture and ligature. Tube. Iodin.
Evacuate.

E. H. 6.
Clavridge, Gilford, 39th Inf., Co. K.
Wd. 10. 10. 18, 6 hr.
Op. 10. 10. 18, 21 h.
1. Wd. by S. F. perforating rt. leg, with fracture of tibia and fibula.
2. Wd. by S. F. penetrating calf rt. leg.
3. Wd. by S. F. tangential, left calf, lower ⅓.
X-ray: fracture of leg, F. B. 10 x 15 mm., 40 mm. deep.
Op., ether: (1) O. E. ant. face middle ⅓. O. S. through Tendo Achillis. Debr., Esq. parc. (2) Extr. of S. F. from wd. of calf. Cabot splint by Lt. Moore. (3) Nettoyage.
Hold.

ABBREVIATION OF FRENCH TERMS

A.C.A. = Ambulance Chirurgicale Automobile ("Autochir").
Abs. = Abstention (no operation done).
Bl. = Blessé (wounded).
Debr. = Débridement.
E.O. = Eclat d'Obus (shell fragment).
Esq. = Esquillectomie (removal of bone fragments, as distinguished from sequestrotomy, which is removal of sequestra).
Ev. = Evacuer (to evacuate).
Exc. = Excision.
Extr. = Extraction.
H.O.E. = Hôpital d'Evacuation.
Nett. = Nettoyage (mechanical cleansing).
O.E. = Orifice d'Entrée (wound of entrance).
O.S. = Orifice de Sortie (wound of exit).
Seton = Superficial perforating wound, not extending beneath deep fascia.
T.S. = Train Sanitaire (hospital train).

ABBREVIATIONS OF ENGLISH TERMS, INCLUDING THOSE RECOMMENDED FOR ADOPTION FROM THE FRENCH

Abs. = Abstention, *i. e.*, examined, and no operation deemed advisable.
A.T.S. = Antitetanic serum.
Ball = Machine-gun or rifle bullet.
Debr. = Débridement, wide opening of wound to make all parts easily accessible.
Esq. = Esquillectomy, removal of bone fragments.
Ev. = Evacuate.
Exc. = Excision.
Extr. = Extraction.
F.S. = Fracture, simple.
F.C.C. = Fracture, compound, comminuted.
M. = Morphin.
Nett. = Nettoyage, mechanical cleansing.
O.E. = Orifice of entry.
O.S. = Orifice of sortie.
S.F. = Shell fragment.

RECEIVING AND OPERATING PAVILIONS FOR A MODERN BASE HOSPITAL[1]

By ASTLEY P. C. ASHHURST, M.D.
PHILADELPHIA, PA.

It is the purpose of this communication to describe the adaptation of standard barrack buildings to the uses of receiving, bathing, operating and *x*-ray pavilions in a base hospital. The buildings described form part of Base Hospital 34, American Expeditionary Forces, which is situated in the outskirts of a large city,[2] at a considerable distance from the front.

It is believed that the use of barrack buildings for these purposes has distinct advantages even when buildings of permanent construction are available. In the case of Base Hospital 34 there is a large, new, modern building of stone and reinforced concrete, which accommodates in itself 1100 beds. It would have been quite possible to have utilized rooms in this building for receiving bathing, operating and *x*-ray departments; but I believe that no building originally constructed for other than hospital purposes will lend itself so admirably as do these barrack buildings to the purposes indicated, since only the four outer walls limit the arrangement of the interior.

The barrack buildings employed at Base Hospital 34 are of what is known as the Fender type, from the name of the manufacturer. The "life" of these buildings is said to be from three to seven years, without noteworthy repairs. They are composed of uniform wooden panels, each 2 meters in width and 2½ meters high, made of two thicknesses of wood, with an intervening air space of 7.5 cm. Each panel, technically speaking, includes the opposite sides of the oblong rectangular building, with the corresponding sections of roof and floor. The roof also is of two layers of wood, separated by an air space, and is covered on the outside with tarred paper. It rises to a peak four meters high and is ventilated about half-way up the slant.

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[1] Recommended for publication by the Chief Surgeon, American Expeditionary Forces. Reprinted from the Transactions of the American Surgical Association, 1918.

[2] Nantes, Loire-Inferieure, France.

The buildings may be made of any desired length, but all are of uniform width of 6 meters. Those in use for various purposes at Base Hospital 34 vary from one which is only 6 panels long (baths for enlisted personnel) up to a number which are 23 panels in length (about 150 feet). These latter serve as wards, with accommodations each for 50 beds.

The rapidity of construction is remarkable. The panels fit together with surprising accuracy, and are fastened by bolts. The enlisted men of the Medical Department, attached to our hospital, learned the mechanism from the French within a very short time, and succeeded in erecting several 15 panel barracks in an average time of one and a half to two days each. This, however, does not include the time necessary to prepare piers for the floors, which may demand two or three days' preliminary work. The piers are made just high enough to bring the buildings level, and their use thus does away with the necessity of grading the ground. If cement floors are employed grading is necessary, and there is also the delay required for the cement to set. But if all the materials are at hand, it should not require more than two weeks to construct one of these pavilions.

The four barrack buildings about to be described are placed in a large level courtyard formed by the three wings of the central building of Base Hospital 34. They are of uniform dimensions, 6 x 30 meters. The building nearest the entrance

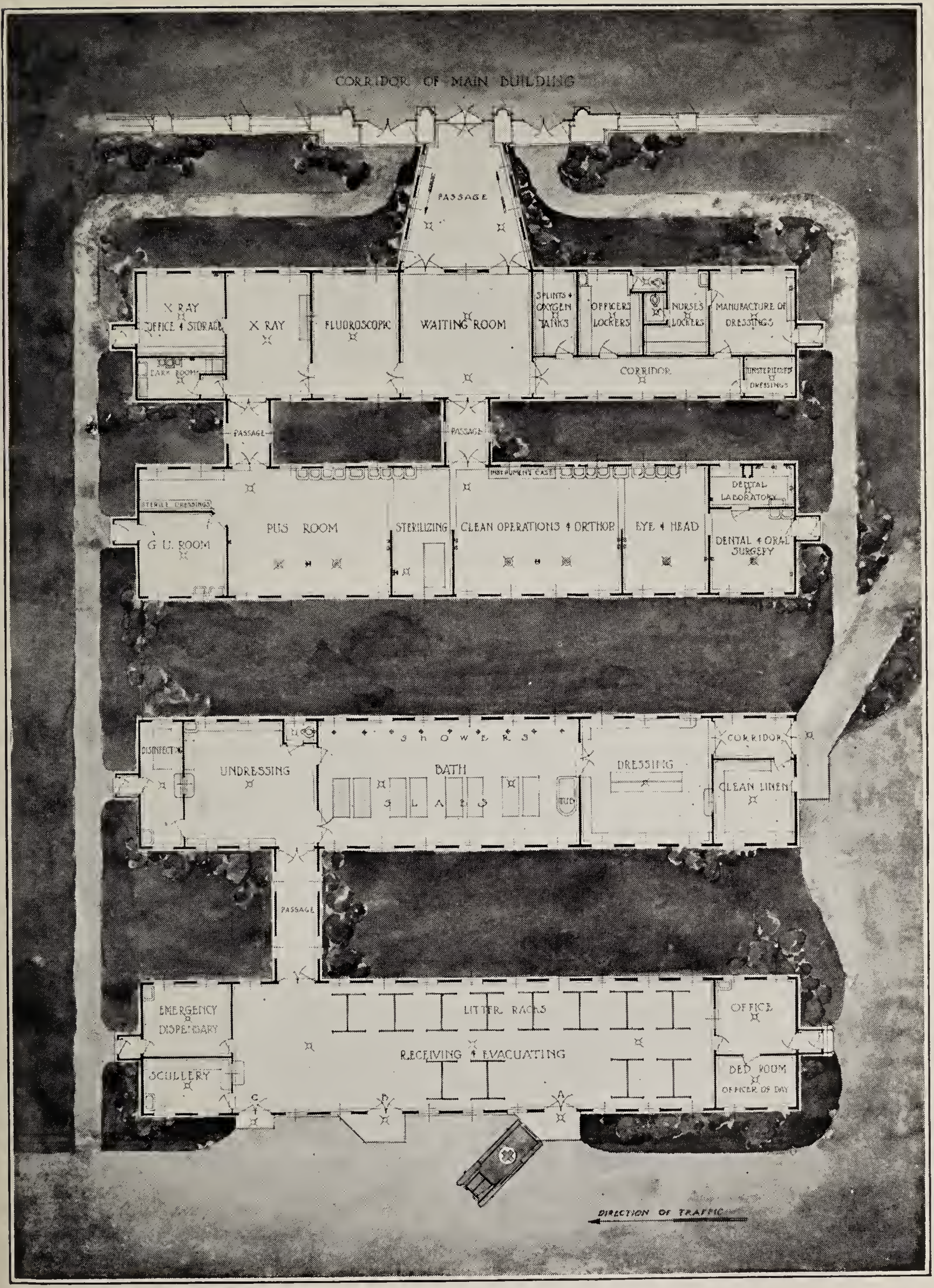

FIG. 7.—Court Buildings, Base Hospital 34, U. S. A., American Expeditionary Forces. Scale, 1 centimeter = 1 meter.

road is the receiving ward; adjoining this, and connected with it by a covered passage, is the bathing pavilion. Parallel with these, but 6 meters distant, is the operating pavilion, while close to this (3 meters distant) is placed the pavilion devoted to *x*-ray laboratories and to dressing rooms for surgeons and for nurses, and a room for the manufacture of surgical dressings. The relations of these buildings are shown in the accompanying plot plan.

The floor of the receiving pavilion is of wood; that of the other three buildings of cement, which is relatively abundant in France, and which certainly makes much more suitable flooring for bathing and operating rooms than does wood. All four buildings are heated by a central hot-water heating system.

As designed by the manufacturers, each alternate panel contains a large window, the intervening panels having small windows placed 2.75 meters from the floor. But as the panels are interchangeable, it was possible in constructing the operating pavilion to place all the large windows on the same side, while beneath the small high windows, thus coming to occupy the opposite side of the building, all plumbing fixtures were conveniently located. A similar arrangement in the bathing pavilion afforded accommodations along the rear wall for a row of shower baths. The floors of the bathing and operating pavilions are constructed with a broad shallow gutter along one side, thus facilitating cleaning and drainage. Owing to the solidity of the floors it was possible to construct all partitions of tile and plaster. The interior of the buildings is painted, that of the operating pavilion being tinted a very pale green. This, with four skylights, gives ample daylight. Artificial light is electric; and gas has been introduced for emergency use.

ADMISSION OF PATIENTS. The incoming ambulances drive up from the east side, and back against the obliquely placed platforms to discharge their loads. By being placed thus obliquely in the road, a clear passage-way is left for other traffic beyond the ambulance; and the ambulances are enabled to continue on their journey westward, after discharge, without the additional backing maneuvers which would be required if the ambulance bodies were stationed at right angles with the road when discharging.

The patients on litters are carried, and ambulatory patients walk, directly into the receiving ward, through doors *A* and *B*, where the litters are placed on racks. The litter bearers at once rejoin their ambulances, passing out by door *C*, and carrying with them fresh litters. Attached to the receiving ward is the office of the officer of the day, as well as his bedroom; at the other end of the ward is a scullery, from which hot coffee may be served, and a minor operating room, in which patients may be catheterized, their dressings temporarily rearranged, and intravenous saline infusions administered. The following regulations are in force:

"A waterproof tag will be attached to the wrist or neck of every patient as soon as he enters the receiving ward. On this tag will be noted the patient's name, the tentative diagnosis, and the ward to which he is assigned. After these data have been thus attached to the patient, the diagnosis tags which accompany him from his last station will be removed and from them will be made the necessary records (Forms 52, 55a and 71 MMD).

"After such emergency treatment as required (Scullery and Minor Operating Room), the patient will be removed to the bathing pavilion by attendants coming thence.

"In the undressing room he will be completely unclothed; only such surgical dressings as are indispensable and the patient's waterproof tag will be retained. His soiled clothes will be listed in duplicate (Form 75, MMD), will be tied loosely in a bundle, the bundle will be tagged (Form 76, MMD), and it will be passed through the window into the disinfecting room. After disinfection they will be turned over to the non-commissioned officer in charge of the storeroom for patient's effects.

"The naked patient will be carried by attendants from the undressing room into the bath room. Patients who can walk may take shower baths. Other patients will be placed on the washing slabs, where they will be thoroughly cleansed with soap and hot water. The hair of the head of all patients, and that of their bodies when necessary, will be closely cropped or shaved. After being dried helpless patients will be carried into the dressing room by clean attendants from that room. The bath attendants will carry patients neither into nor out of the bath room.

"In the dressing room the patients will be given hospital bedclothing, and will be carried to the proper ward by ward attendants. On leaving the dressing room the non-commissioned officer from the receiving ward will see that Form 55a (the "Clinical Brief" of the history) is attached to the proper patient, using the waterproof tag for identification."

The clothing of incoming patients is disinfected in a steam sterilizer, adjacent to the undressing room in the bathing pavilion. The washing of the patients is much facilitated by the use of slabs instead of bathtubs, though one tub is provided. These slabs are of slate, draining to a deep gutter on all sides, and the gutter draining at the foot of the table, the frame of which is constructed of iron piping. It is only when they are placed on the washing slabs that the patients are first moved from the litters on which they have been transported. The soiled litters are cleaned, and are carried back into the receiving ward when the attendants in the undressing room make one of their trips into that ward for other patients.

THE OPERATING AND *X*-RAY PAVILIONS. These are only 3 meters apart, and being joined by two wide passages form almost one continuous building. They are approached through a wide covered passage-way leading from a recovery room in the center of the main building. Opening out of this recovery room are two surgical wards, with accommodations for 75 beds; while a barrack ward, of 50 beds capacity, is accessible only 3 meters distant on the other side of the main building. Thus there are available, on the ground level, 125 beds for patients recently subjected to operation, where they may be kept until it is safe for them to be removed to more distant wards.

In the *x*-ray department are fluoroscopic and radiographic rooms, an office, and the dark-room. These rooms connect by a short wide passage with the operating rooms.

The other end of this *x*-ray pavilion is devoted to dressing rooms for surgeons and nurses, and a room for the preparation of dressings for sterilization. As the dressings come to us already made, through the intermediary of the Red Cross, it is necessary for our nurses only to sort the ready-made dressings into packages suitable for sterilization. Until they are needed for sterilization the dressings are stored in a closet next the nurses' workroom. There is also a large closet for storage of blankets, splints, oxygen tanks, plaster of Paris, etc.

There are several well-lighted rooms available for purposes of anesthetizing patients; this may be done in the wide passageway leading from the recovery room to the waiting room, in the waiting room itself (when not otherwise occupied), in the corridor to the east of the waiting room, or in one of the operating alcoves not in actual use.

In the operating pavilion proper there are accommodations for seven simultaneous operations—a limit which is not likely often to be exceeded. Specifically there are accommodations for oral surgery, including dental operations, with a well appointed dental laboratory for manufacture of prostheses immediately adjacent; an alcove for head surgery in general, including also the eye, ear, nose, and throat; an alcove to accommodate two tables for orthopedic and other aseptic operations; another accommodating two tables for septic cases; and a room for genito-urinary surgery. The steam sterilizing apparatus is placed near the center of the pavilion, and ample accommodations are provided for storing sterile dressings near at hand.

In planning this operating pavilion it was determined to attempt a happy medium between the grand saloon type of operating room, where there are no partitions whatever, and the type where each operating table has its special room. The latter plan lacks economy in administration, many more attendants are required, and time and labor are lost by walking around so many corners instead of going directly to the place desired. The former plan, where numerous operators work simultaneously in the same large room, does not always tend to quiet and discipline, and injurious draughts may arise. By employing partitions 3 meters long (which reach half across a barrack building of this type), it was possible to form alcoves of sufficient depth to afford relative isolation for the operating teams; and by arranging that the two alcoves in most constant use should each be of sufficient size to accommodate two tables when necessary, it became possible to reduce to the minimum the number of attendants required, as one sterile-handed nurse may easily conduct a suture table for two simultaneous operations.

My thanks are due to many members of our professional staff for suggestions of value in connection with the construction of these buildings, and especially to the Chief Engineer Officer, Major Fowler, for procuring material and giving us opportunity to use it as we deemed best. I have also to express indebtedness to Corporal Hoke and Private Stern for their architectural knowledge and for drawing numerous plans.

CASES ILLUSTRATING GUNSHOT WOUNDS OF THE VASCULAR SYSTEM[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

Dr. Astley P. C. Ashhurst reported the following cases illustrating gunshot wounds of the vascular system:

Case I.—*Dry Lesion of the Axillary Artery from Bullet wound; death sixteen hours after ligation.*

Tony Pulchi, aged twenty-six years, was admitted to the Episcopal Hospital December 31, 1916, immediately after having been shot, while an innocent bystander in a street fight. The wound of entrance was at the posterior edge of the right deltoid, close to its humeral insertion, and there was no wound of exit. *X*-rays localized the bullet lodged against the second right rib. His radial pulse was equal on both sides, and apart from slight pain and swelling in the axilla there were no serious symptoms. On the day after admission, however, weakness of the muscles supplied by the median nerve and paralysis of those supplied by the musculospiral nerve were noted. It was determined to explore the axilla to ascertain the nature of the nerve lesions.

Operation, January 5, 1917, by Dr. Ashhurst. Ether anesthesia. An incision was made from the middle of the claxicle downward and outward, in the space between the deltoid and pectoralis major, exposing the pectoralis minor. A finger was then passed under the latter muscle, preparatory to its division, for exposure of the axillary plexus. As soon as the finger emerged at the lower border of the pectoralis minor a gush of arterial blood burst through at both borders of the muscle. It was evident that the bullet had injured the axillary artery and the primary bleeding had been checked by a clot or that a slough in the wall of the artery had been separated only when the finger entered the axilla. The profuse hemorrhage was checked temporarily by the operator compressing the axillary artery with his left finger just below the clavicle and with his right finger below the pectoralis minor. Removal of either finger released a perfect flood of arterial blood. Dr. Spruance, who was assisting in the operation, was then entrusted with digital control of the distal end of the artery, thus releasing the operator's right hand. Attempts were then made to clamp the artery above the lesion, but these proved ineffectual, owing to the depth of the wound and the inability to distinguish the structures. Dr. Spruance then compressed the subclavian against the first rib, controlling hemorrhage from the proximal end of the axillary, while the operator compressed the distal end and tried to clamp it; this also proved ineffectual at first, it being impossible to clamp the artery without pinching one or other of the nerve trunks; but finally the clamp was properly placed and the distal end ceased to bleed. Dr. Spruance then compressed the axillary just below the clavicle, while Dr. Ashhurst ligated (with a double strand of No. 2 chromic catgut) the third portion of the subclavian by the usual incision above the clavicle. This at once stopped the pulse at the wrist. After suturing the cervical incision the axillary wound was again exposed and found to be dry. The axillary incision was then enlarged, dividing the tendons of the pectoralis major and minor. Removal of the hemostats still in the axillary wound then was begun; removal of the last hemostat was again followed by profuse hemorrhage, which was not controlled by digital compression of the axillary below the bleeding-point. It was now found that this profuse hemorrhage came as recurrent bleeding from the subscapular artery, there being a bullet hole in the axillary just opposite the origin of the subscapular (Fig. 8.). Therefore the axillary artery was tied above and below the hole and the subscapular artery was tied also. The axillary plexus of nerves was then examined: the median nerve had been bruised by hemostats; the musculospiral nerve was contused, presumably by the bullet; the ulnar and musculocutaneous nerves were undamaged. The divided muscles were repaired and the wound closed. The patient was in a precarious condition as the result of hemorrhage and died ten hours after operation, in spite of stimulation. It is possible that blood transfusion might have saved his life, but no donor was available.

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[1] Read before the Philadelphia Academy of Surgery, October 6, 1919. Reprinted from Annals of Surgery, 1920, i, 98.

Case II.—*Recent bullet wound of the right axillary artery, with diffuse traumatic aneurysm; ligation of first portion of subclavian artery and of axillary artery above and below the wound; recovery.*

Charles W., a private of the 104th Infantry, U. S. A., was hit by a machine-gun bullet in the Argonne Forest, November 9, 1918, at 4 A.M. He was brought to Evacuation Hospital No. 6 and operated on twenty hours later. Fluoroscopic examination by Capt. Angell showed the bullet superficially placed in the right pectoral region. The wound of entrance was over the right shoulder posteriorly. There was an immense pulsating hematoma occupying the entire right pectoral region, which was discolored by the extravasated blood. The bullet was palpable just beneath the skin and the hematoma appeared on the point of rupture. There was no pulse at the right wrist.

Under ether anesthesia, immediate operation was undertaken, with the skilful assistance of Capt. Morse and Lieut. H. S. Kerchner. It was determined to do preliminary ligation of the first portion of the subclavian, as the immense size of the hematoma rendered the third portion inaccessible through healthy tissues, and then to expose the axillary artery where wounded. Accordingly, an incision was made along the inner end of the right clavicle and downward for 7 cm. over the sternum and the inner end of the clavicle (2.5 cm.) was resected. The pleura was accidentally punctured. The first portion of the subclavian artery was then exposed and the pneumogastric nerve and its recurrent laryngeal branch were identified (Fig. 9.) Owing to the large size of the hematoma these structures lay at an abnormal depth. A ligature of No. 2 chromic catgut was passed around the artery distal to the nerve. On tying this ligature, pulsation in the hematoma stopped at once and the mass decreased in size. The operative incision was closed in layers without drainage. *A second incision was now made in the line of the axillary artery from the clavicle to the anterior axillary fold, dividing the pectoralis major and minor muscles.* The bullet was removed from beneath the skin and the clots evacuated. Free arterial bleeding then occurred from the proximal end of the axillary artery just distal to the clavicle. This was checked by the finger and then by hemostatic forceps, when it was seen that the bullet had clearly and completely divided the first portion of the axillary artery without injuring the vein (Fig. 10). Both ends of the artery were ligated with No. 2 chromic gut and the wound was left wide open and drained by rubber tissue. It was directed that the patient be not evacuated.

The next day the patient's hand was warm and the circulation appeared to be reëstablished. He could flex his fingers and the axillary plexus of nerves apparently had escaped injury. He has been very hoarse since the operation, perhaps from operative injury

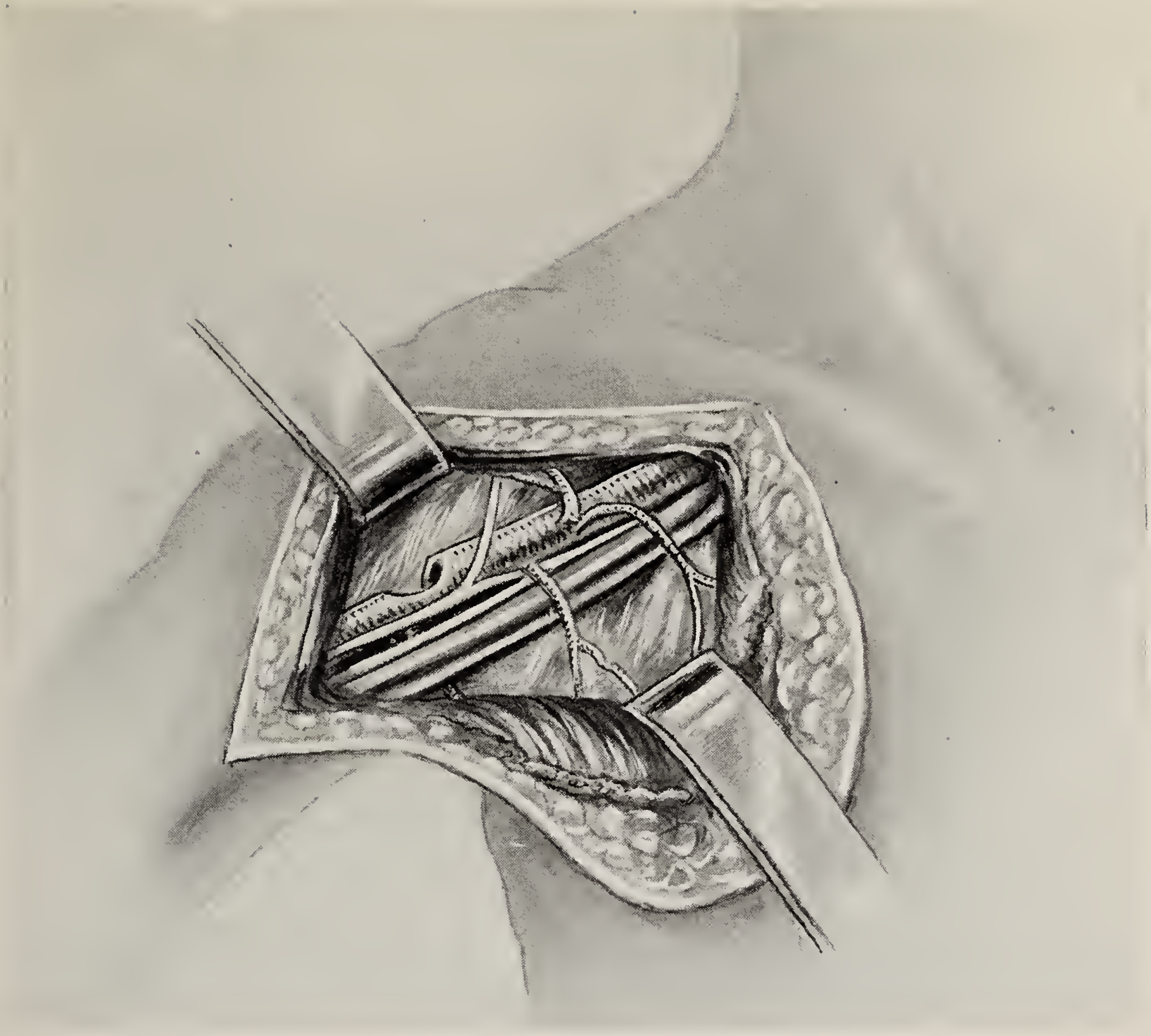

FIG. 8.—Case 1. Bullet wound of axillary artery.

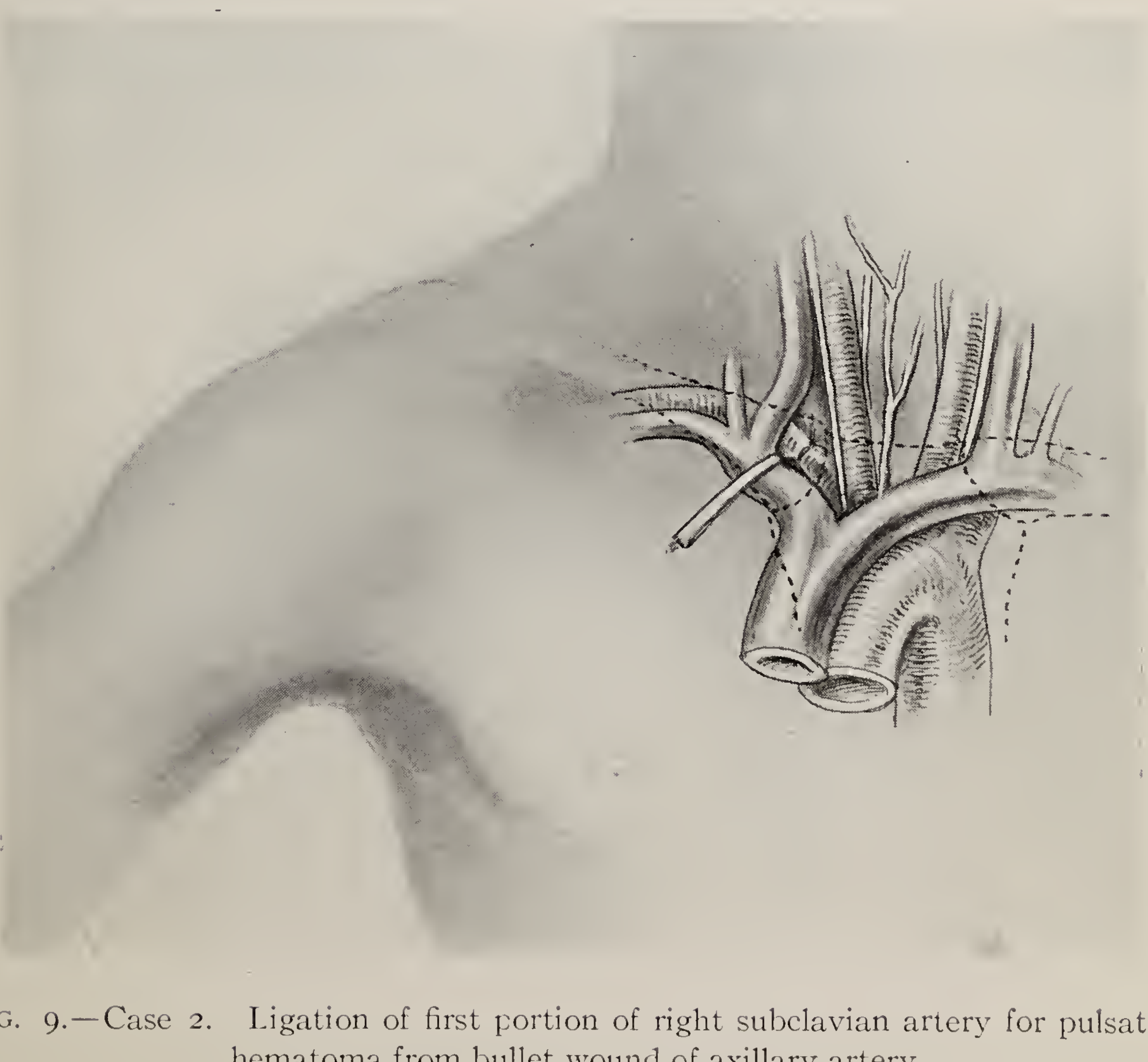

FIG. 9.—Case 2. Ligation of first portion of right subclavian artery for pulsating hematoma from bullet wound of axillary artery.

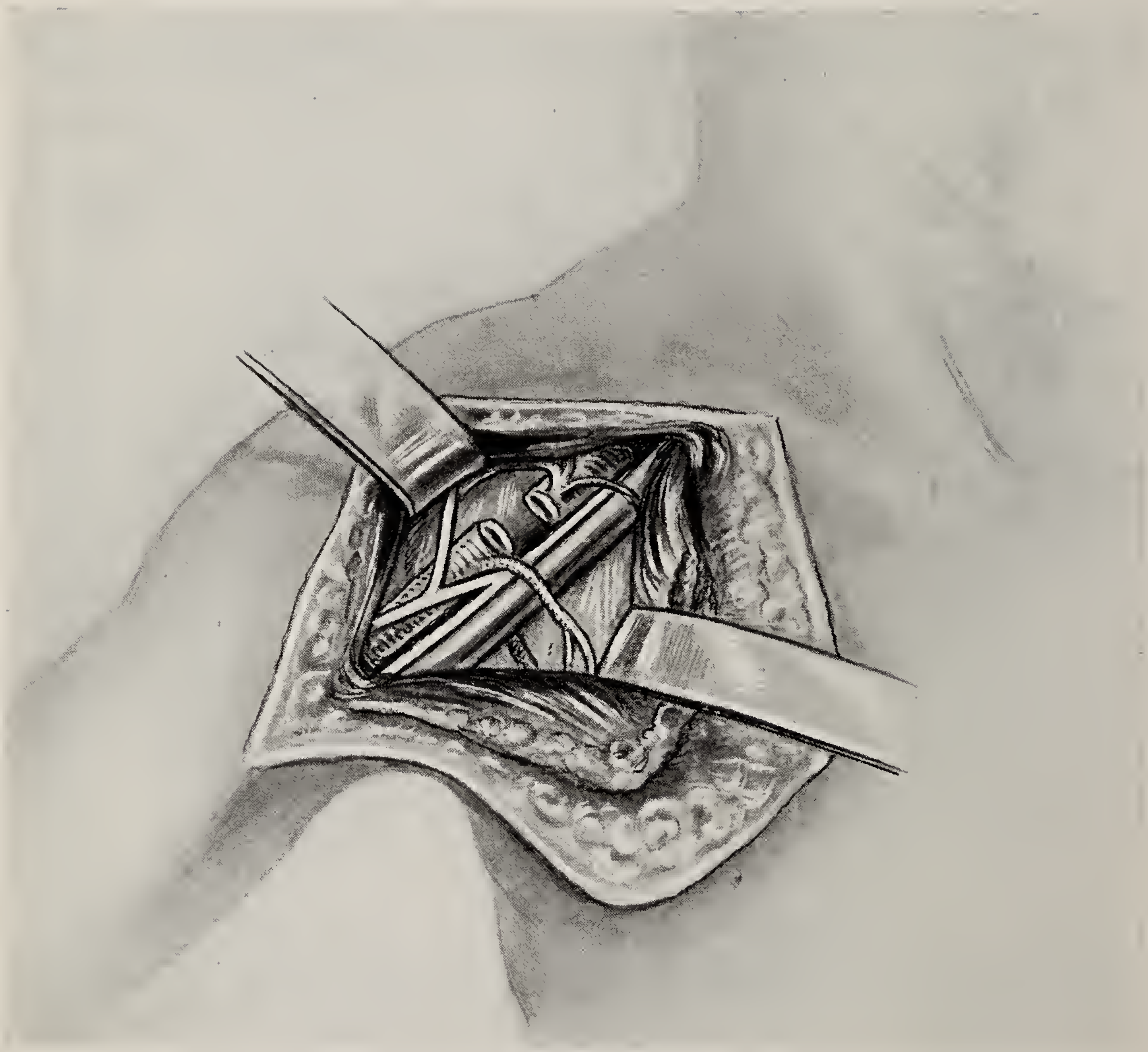

FIG. 10.—Case 2. Axillary artery severed by bullet.

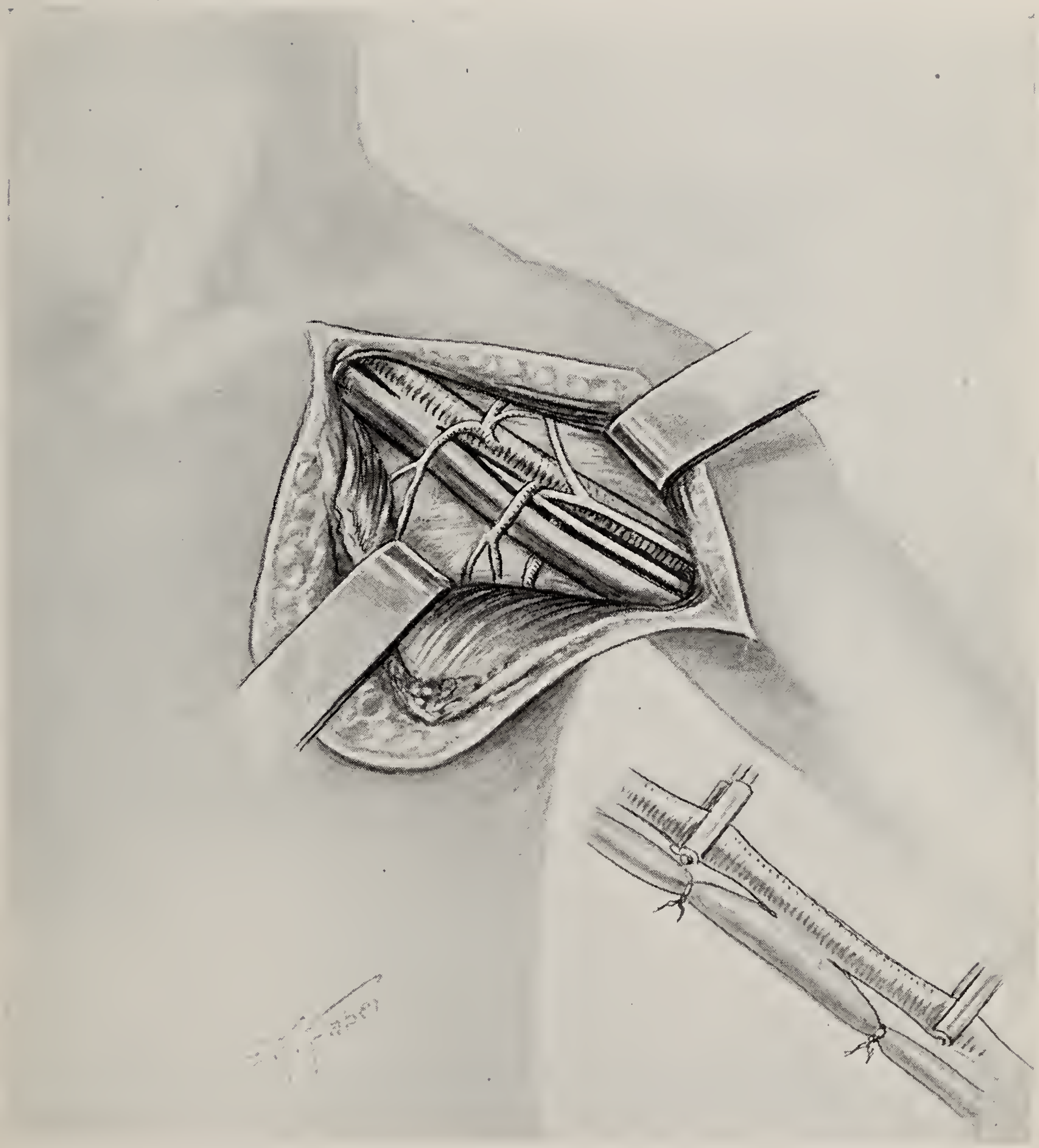

FIG. 11.—Case 3. Direct arteriovenous fistula of first part of axillary artery.

of the recurrent laryngeal nerve. The wounds were dressed: the sternal wound was healthy, but the pectoral wound smelled of gas-gangrene, and a smear showed the presence of the B. aërogenes capsulatus. It is to be remembered that the tissues in the axilla were widely lacerated by the escaping blood and had been under increasing pressure from the hematoma for twenty hours before operation, and after operation were still deprived of their normal blood supply. At the time of operation the patient's condition did not warrant an excision of all the muscles which were infiltrated with blood. French anti-gas-gangrene serum was administered and the wound was treated with Dakin's solution according to Carrel's technic.

On the third day the patient was still very hoarse, but the pectoral wound looked better, though the muscles were still very spongy. It was noted that he was unable to extend his fingers or wrist, but it was not determined whether this was due to weakness or to a nerve lesion.

Five days after operation the patient appeared convalescent; though he was still very hoarse, and had a little cough, the wound was doing very well.

Six days after operation he was evacuated in good condition; the sternal wound had remained clean and the pectoral wound was doing well.

Inquiries of the War Department as to the patient's subsequent history have been unanswered.

CASE III.—*Direct arteriovenous fistula of the axillary vessels following wound by shell fragment. Excision of vein, suture of artery; recovery.*

George W., private, 39th Infantry, U. S. A., sustained a penetrating wound by a shell fragment August 1, 1918, in France. The wound of entrance was in the left deltoid region. He was taken to a hospital and put to bed. No operation was done. He did well, presenting no noteworthy symptoms. After a few days he got out of bed, and soon began to work about the ward. One night (about 2 A.M.), eight or ten days after injury, he was awakened by pain in the left subclavicular region and down the left chest. Examination by the ward surgeon revealed a blowing murmur in the left subclavicular region. Previous examinations, he said, had shown a heart murmur but nothing abnormal in the axillary region.

This patient first came under Dr. Ashhurst's notice seven months after his injury, in the Walter Reed General Hospital, Washington, D. C., where he was in the service of Major E. M. Jones, who asked Dr. Ashhurst to operate. The disability consisted in

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inability to raise the arm above the head, this motion being only two-thirds of normal. There was a buzzing and whirring on palpation below the left clavicle, but the radial pulse on the two limbs was equal and synchronous and the blood-pressure was normal on both sides. Skiagraphic examination showed a shell fragment 3 cm. anterior to the vertebral end of the fourth left rib. This fragment, of medium size, was producing no symptoms and its removal was not indicated.

On March 27, 1919, with the skilful assistance of Major Jones and Lieut. J. C. Lawler, Col. Ashhurst operated under ether anesthesia; as the site of the lesion evidently was high in the axilla, it was determined first to control the circulation by clamping the third portion of the subclavian artery, which was done through the classical incision. Next an incision was made convex toward the greater tuberosity of the humerus, from the middle of the clavicle to the anterior axillary fold near the chest wall. After ligating the cephalic vein the tendon of the pectoralis major was divided, and the axillary artery was clamped just proximal to the origin of the subscapular artery. The tendon of the pectoralis minor was then cut and the clavicular origin of the pectoralis major divided for about 3 cm. The lesion could then be identified, after dissection of scar tissue, as a direct arteriovenous fistula of the axillary vessels, involving the first portion of the axillary artery (Fig. 11). The *outside diameter of the fistula was about* 1 *cm.* The axillary vein was carefully cleared, ligated above and below the lesion, cut across between the respective ligatures and the lesion and dissected free as a pouch attached to the artery. Artery clamps were now applied just proximal and just distal to the lesion, and the clamps which for about one hour had been on the subclavian and on the third portion of the axillary were removed. A grooved director, passed across the venous pouch through the fistula into the lumen of the artery, demonstrated completely the nature of the lesion. Three interrupted sutures of fine linen were now passed through the arterial wall at the site of the fistulous opening and were tied after cutting away the venous pouch from the artery. Then a continuous through-and-through linen suture was applied to the artery and the arterial clamps were removed. There was absolutely no leakage from the suture line as the artery filled out and pulsated, but a branch of the acromiothoracic artery below the lesion spurted actively and was tied. The pectoralis major (not the minor) and the skin were closed separately with chromic gut, a small tube being left at the lower angle of the large wound. The neck wound was closed without drainage. The duration of the operation was three hours.

Recovery was uneventful, no abnormal symptoms of any kind being observed, and function of the arm being gradually restored. The patient returned to his home and has recently been carefully examined by Dr. Lucian H. Landry, of New Orleans, one of Dr. Matas's associates, who courteously wrote September 15, 1919, that after a careful examination he considered the result a complete cure.

Postscript. At the meeting of the American Surgical Association, in St. Louis, May, 1920, Dr. A. E. Halsted, of Chicago, informed Dr. Ashhurst that this patient had recently come under his observation, with a recurrence of symptoms, which followed immediately upon very violent abduction of the shoulder while performing a snake-dance with some boisterous companions.

CASE IV.—*Circumscribed traumatic aneurysm of sural artery following wound by shell fragment; obliterative endo-aneurysmorrhaphy; recovery.*

Richard B., 1st Lieut., U. S. A., aged twenty-four years, suffered a perforating wound of the right popliteal space by a minute shell fragment September 26, 1918. The punctured wounds of entry and exit healed without any operative treatment, but after some weeks, while walking, he felt a sudden sharp pain in the bend of the knee and a pulsating tumor appeared. He came under Dr. Ashhurst's notice more than five months later, in March, 1919, at the Walter Reed General Hospital. Low in the right popliteal space was a tense, hard, not tender swelling, 7 by 5 cm., its long axis corresponding to that of the limb. This swelling exhibited distinct expansile pulsation and bruit. There was no appreciable difference in the pulse below the knee in either leg.

Operation was undertaken March 6, 1919. Under Esmarch anemia a longitudinal incision, 16 cm. in length, was made over the popliteal space, and the deep fascia and heads of the gastrocnemius muscle, as well as the internal popliteal (posterior tibial) nerve were dissected off the sac, all of these structures being densely adherent. The sac was opened on its median side and some well-organized clots were evacuated. The sac was found to be of the size of a large hen's egg: its walls were formed of organized granulation tissue, except for an area about 3 by 2 cm. on the median and ventral surfaces, which was white and glistening, representing the original intima of the wounded artery, now spread out to form part of the sac wall. This patch of typical intima contained two minute orifices—one proximal and the other distal—about 2.75 cm. apart, evidently representing the afferent and efferent channels for the blood. The proximal opening bled a little. There were no other openings in the sac. Most of the posterior wall of the sac was dissected free and excised; the arterial orifices were closed by linen sutures; and the remainder of the sac, which was too adherent to be excised easily, was obliterated by No. 000 chromic gut sutures. The fascia and skin were closed separately. The time of the operation was about one hour.

Recovery was uneventful, all the symptoms being relieved.

EXPERIMENTAL IMPLANTATION OF FOREIGN TISSUE INTO THE LUMEN OF LARGE ARTERIES[1]

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HAVING been interested by Herrick's[2] report of implanting musculofascial strips into the lumen of arteries without subsequent clotting, the following experiments were made by Capt. Dozier, Capt. Propst and myself while on the surgical service of Evacuation Hospital No. 36, A. E. F., France, to determine if our experiments would substantiate Herrick's findings.

EXPERIMENT 1. March 12, 1919. A young mongrel bitch, weight 11 kg., was anesthetized with morphin and ether. The abdomen was opened by a 7.5 cm. incision in the midline, the intestines pushed to the side, the right common iliac artery isolated and its blood current arrested by two small clamps placed about 2 cm. apart. A fine silk suture was passed through the center of the artery by means of a small cutting needle, and by means of traction on this suture a small cataract knife was drawn through, making a slit about 3 mm. long through each side of the artery. The knife was withdrawn and by means of the same suture a strip of muscle and fascia from the rectus abdominis, one-half the diameter of the lumen of the artery, was drawn through the slits, projecting on each side about 4 mm. A suture on each side was required to control a slight bleeding proximal to the insert. The clamps were removed and the pulse below was of decidedly less volume than on the opposite side. The abdomen was closed in three layers. The animal made a rapid and uneventful recovery from the operation.

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[1] Reprinted from the Journal of the American Medical Association, 1920, lxxv, 737.

[2] An Experimental Study of the Surgery of Large Arteries, Jour. Am. Med. Assn., December 28, 1918, lxxi, 2120; Camp Crane, Allentown, Pa.

Necropsy. June 2, 1919. The animal was anesthetized and the abdomen opened. The pulse was apparently of equal volume in each femoral. Except for some adhesions the artery presented no external evidence of having been molested. One cm. of the artery was resected and on being held to the light presented a perfectly smooth and regular lumen, with a band about one-quarter its diameter crossing its center from side to side. On examination this band was found to be smooth and glistening and apparently covered with endothelium. There were no clots adherent to it.

Experiment 2. March 18, 1919. Young male dog, weight 6 kg. Under ether and morphin anesthesia the right common carotid was isolated and a musculofascial strip, from the sternomastoid, one-half the diameter of the lumen of the artery, was passed transversely through the center of the vessel as described in Experiment 1. There was no bleeding after pulling this insert through. The pulse in the carotid above this was very weak. The wound was closed and the dog made an uneventful recovery.

Necropsy. June 2, 1919. Under ether anesthesia the carotid was exposed and 1 cm. of it was removed. There was a small teat of tissue projecting from one side of the artery, the only indication of the operation on viewing it from the outside, but across its lumen passed a smooth glistening band of tissue about one-quarter the diameter of the lumen of the artery.

Experiment 3. March 23, 1919. Young male dog, weight 6 kg., was anesthetized with morphin and ether, and the right common carotid isolated and clamped. Then two-thirds of its circumference was removed for a distance of 1 cm. Into this space was sutured a piece of fascia and muscle 4 mm. thick, taken from the sternomastoid. After releasing the clamps there was a barely perceptible pulse above the operated area. The wound was closed in the usual manner.

Necropsy. June 2, 1919. On opening the neck through the old scar it was found that the artery was replaced by a small fibrous cord for a distance of about 2 cm. There was no lumen to the cord. This was probably caused by suturing in the graft too closely, not leaving sufficient room for the blood current.

Experiment 4. April 8, 1919. The same animal used for Experiment 1 was anesthetized and the right common carotid isolated in the usual manner; a musculofascial insert of approximately half the diameter of the lumen of the vessel was then placed as in the first two experiments.

Necropsy. June 2, 1919. On exposing the carotid a few adhesions were found; $1\frac{1}{2}$ cm. of the vessel were removed. A very minute smooth glistening cord passed transversely through the center of this section. It was hardly 1 mm. in diameter. There was no clotting about the insert.

EXPERIMENT 5. April 24, 1919. Mongrel bitch, weight 12 kg. An insert one-half the diameter of the lumen of the vessel was placed transversely in the left common iliac artery.

Necropsy. June 3, 1919. In this case the cord was smooth and glistening and about one-third the diameter of the artery lumen. It was apparently covered with endothelium and there were no clots adherent to it.

EXPERIMENT 6. April 29, 1919. Mongrel bitch, weight, 11 kg. Right common carotid was exposed and one-third of its circumference was removed for a distance of 7 mm. A piece of sternomastoid muscle and fascia 4 mm. thick was removed and sutured as a patch over the carotid defect, with the muscle surface turned inward.

Necropsy. June 3, 1919. There were rather dense adhesions about the patch, but the artery was isolated with little difficulty. The pulse on this side was very good, but probably of a little less volume than the opposite side. A section of the artery was removed and slit open. The patch was projecting into the lumen of the vessel for about 1 mm., flattening that side of the lumen to a slight extent. It was smooth, firm and glistening. No clots were present. The graft did not show any signs of beginning aneurysmal bulging, though the autopsy was performed thirty-five days after the graft was placed.

EXPERIMENTS 7 and 8, May 1, 1919. Experiment 9, May 2, 1919. Experiment 10, May 4, 1919, and Experiment 11, May 5, 1919. These experiments were all performed in the same manner, using the common carotid artery. In every case we tried to make the musculofascial insert as near one-half the diameter of the lumen of the artery as possible. In none of the experiments was it necessary to take a stitch in the edges of the slit through which the insert was passed, in order to stop bleeding, as the insert fitted tightly and the elasticity of the vessel wall made it close firmly about it. The necropsies were all done June 3, 1919, and in every case there was a good pulse distal to the insert. No clots were present. All the inserts were smooth, glistening fibrous bands of one-third to one-sixth the diameter of the artery lumen.

This report is incomplete in that no histological examinations were made. Soon after making the autopsies we received orders to move and the specimens were lost in moving. But I think the foregoing work substantiates Herrick's findings and justifies two very important conclusions: First, that a foreign tissue can be placed in the lumen of an artery without immediate absorption and without the formation of a permanent clot; second, vessel walls can be sutured without necessarily having intima to intima approximation. Further work on this interesting subject will show whether or not a temporary clot is usually formed and whether the insert is ultimately absorbed.

PLASTIC (RECONSTRUCTIVE) SURGERY OF THE HAND AND THE FOREARM[1]

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Reconstructive surgery of the hand and the forearm is of very great importance because of the large number of mechanics and workmen who suffer injury to these members and who are more or less permanently disabled thereby. Injuries of the upper extremities are much more disabling than similar injuries of the lower extremities.

It is difficult surgery because of the complex and fine structure and function of the hand. There are certain principles which must be here strictly observed; for ill-advised, poorly-planned, and improperly-executed operations will only make the patient worse. And even slight infections may destroy your finest work.

Finally, this branch of surgery has been more or less neglected by the general surgeon. It is often tedious and requires considerable patience and prolonged after-treatment. Unfortunately, repair of injuries of the hand and the forearm is frequently made by the inexperienced hospital interne in the receiving ward. The patient is afterward dressed in the surgical dispensary. And as his wound heals he is discharged with a more or less disabled hand. He departs, taking it for granted that nothing more can be done for him. Seven of the following eleven cases had hospital treatment before they were seen by the author.

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[1] Read before the Philadelphia Academy of Surgery, February 7, 1918. Reprinted from Annals of Surgery, July, 1918.

The principles of orthopedic surgery applied in the operative work and in the after-treatment by massage, electricity, hydrotherapy, active and passive movements, and by the use of special splints as necessary, will go far toward the restoration of these industrial cripples to their former positions of usefulness and independence.

The following cases are presented to illustrate some of these methods of surgery and their results:

Case I.—E. M. In August, 1914, she had an infection of the right hand. Sinuses remained open at the sites of incision for five weeks. The fourth and fifth fingers were left stiff in almost complete extension, while active and passive flexion were impossible. Massage was begun and continued for almost eight months with but little improvement. She was then referred to the orthopedic clinic at the Presbyterian Hospital. Examination showed that the extensor tendons of the fourth and fifth fingers were adherent on the dorsum of the hand, that the fingers were held in almost full extension, and that active and passive flexion of these fingers was greatly limited and painful.

Operation (May 3, 1915). Through a longitudinal incision the extensor tendons of the fourth and fifth fingers were exposed over their entire course on the dorsum of the hand. They were found firmly adherent to the skin and to the structures beneath them. The adhesions were divided with a sharp knife to cause as little injury to the tissues as possible. The fingers could then be well flexed passively. The incision was sutured. The wound healed by first intention. Ten days later massage was begun. Some improvement followed this operation, but active and passive flexion was incomplete and forced motion or active effort elicited fairly severe pain about the metacarpophalangeal joints.

Second Operation (October 5, 1915). The tendons were freed from adhesions and were surrounded by a layer of fat taken from the thigh. Improvement following this operation was rapid, and within a few months she regained complete normal motion and function of the two fingers. A good result was obtained only when the tendons were surrounded by fat from the thigh.

Case II.—M. N. Seen at the Orthopædic Hospital on May 26, 1917. In August, 1916, her left hand had been cut with glass on the ulnar side of the palm. An operation had been performed at another hospital in Philadelphia. Examination showed a long dense scar extending longitudinally on the ulnar side of the palmar surface of the left hand. The little finger was held in a position of partial flexion beyond which active and passive extension were impossible. Passive flexion was complete, but active flexion was absent. There was numbness over the two distal phalanges. The position of the finger interfered with her work in the mill.

Operation (September 7, 1917). Incision along the old scar. After considerable dissection the ends of the severed flexor tendons were found buried in scar tissue one and one-half inches apart. The finger could not be fully extended owing to contracture in the joints. Union of the separated ends of the tendons was secured by means of a bridge of fibrous tissue that was adherent to the distal ends. A piece of fat from the thigh was placed about the tendon, and the wound was closed.

September 22, 1917. Wound healed, no active motion of sutured tendons.

October 23, 1917. Good active motion in metacarpophalangeal joint, none in the interphalangeal joints.

November 17, 1917. Moderate contracture in interphalangeal joints. Slight active flexion of second and third phalanges. Sharp, cutting pain in palm on motion of finger, tendons slightly adherent to scar in region of distal transverse palmar crease.

February 23, 1918. On making fist the finger flexes completely into the palm; but on holding the other fingers extended the little finger does not flex completely. Active flexion in all joints. Passive extension incomplete, even when metacarpophalangeal joint is flexed. Complete return of sensation in finger.

March 30, 1918. Good active flexion in all joints. Passive and active extension incomplete and identical. Good function of finger, no interference with work; but patient complains of pain in distal palmar crease on flexion of the finger. Advise second operation with incision along line of palmar crease to free tendons from adhesions and to place another small layer of fat from the thigh about them.

Discussion. When operating on this case the mistake was made in making a longitudinal incision along the line of the old incision in the palm. A transverse incision along the distal palmar crease and then longitudinally along the ulnar side of the hand would have been better. The original longitudinal incision of the palm left a hard adherent scar a year later, and a thick firm scar is still present from the last operation. A longitudinal incision on the dorsum of the hand leaves no bad results, as illustrated in Case I, but on the palmar surface of the hand and fingers it should always be avoided if possible. The following cases illustrate the advantage of a transverse incision along the line of a natural crease.

Case III.—G. H., a mechanic. September 20, 1917, he was struck on the palmar surface of the wrist by a steel shaving. The foreman of the shop drew out a piece of steel which was protruding from the skin. The wound soon healed.

October 16, 1917. Referred to my dispensary at the Presbyterian Hospital. Diminished sensation present along the median nerve distribution. The nerve evidently had been injured in the wrist by the steel. Unable to make good fist. Very little power in fourth and fifth fingers in active flexion.

January 8, 1918. Almost complete return of normal sensation. Only one-half normal active flexion of fourth and fifth fingers. A small firm mass felt in palm along line of their tendons. An *x*-ray plate showed a long narrow piece of steel present in palm of hand.

Operation (January 15, 1918). Incision along distal palmar crease. After a fairly long search the steel was found within the common sheath of the flexor tendons lying between the superficial and the deep. Wound closed with three fine catgut sutures.

February 7, 1918. Wound healed, scar scarcely visible. Patient has returned to work. Complete normal function of fingers and hand.

Case IV.—L. S. December 22, 1917. Orthopædic Hospital. In September a heavy door closed on his left hand, and the ring which he was wearing on his fourth finger severed the flexor tendons of the finger. Operation on the finger was performed in Pittsburgh a month ago. On examination there is present a linear scar on the palmar surface of the ring finger extending from the middle of the second phalanx to the metacarpophalangeal joint. There is complete active flexion of the metacarpophalangeal joint, but none in the interphalangeal joints. The tendons are adherent to the proximal end of the scar. Finger remains in full extension and passive flexion is normal.

Operation (January 25, 1918). Incision along the old scar. The sublimis and the profundus tendons were found adherent to the scar in its entire extent, but not severed. A fine layer of fat was taken from the lower part of the external surface of the thigh next to the fascia lata. This fat was sutured about the tendons, and a pulley was formed at the proximal interphalangeal joint for the profundus tendon. Fine chromic gut (00) threaded on fine needles was used for this work. Wound closed. Two days after operation the patient became ill and had a high fever. Fearing that there might be a streptococcic infection of the finger I opened the wound. Only a little serum was present, but the wound was allowed to remain open. Within twelve hours the patient developed well-defined scarlet fever and was at once transferred to the Municipal Hospital. While there the fat sloughed out of the finger and the wound healed.

March 24, 1918. Wound healed. No active flexion of the finger. Tendons are adherent to the scar. Advise second operation when the general condition of the patient is normal again.

Discussion. A tedious, delicate operation was rendered useless by a slight infection which occurred either at the time of operation or when the incision was reopened. A flap incision along the side of the finger and across the crease at the distal interphalangeal joint would probably have been better than the incision along the old scar on the palmar surface of the finger. The fat which can be used in the finger must be very delicate. The coarse subcutaneous fat from the thigh, particularly in women, is not suitable. In patients who are slender, the inner portion of the fat next to the fascia lata may be employed. This fat is covered with a delicate membrane which may well make a suitable tendon sheath. The tissue about a hamstring tendon or along the tendo Achillis, as advised by Bunnell, should give a good result. A pulley should be made for the flexor tendons where they span a joint.

The following two cases illustrate the suturing of tendons of the fingers under local anesthesia:

Case V.—E. B., a librarian, November 5, 1917. A month ago she cut her left thumb with a steel paper-knife. The incision was small and deep and it divided the tendon of the extensor longus pollicis just distally to the metacarpophalangeal joint. The second phalanx of the thumb remains in partial flexion and no active extension is possible.

Operation (November 8, 1917). Under local anesthesia. The two ends of the tendon were dissected free from scar tissue and sutured with fine chromic gut. Patient could then flex and extend thumb to the normal limit. Wound closed. Uneventful recovery with good function except for slight adhesion of skin at site of incision.

Case VI.—A. B., a machinist. Seen at the Episcopal Hospital. October 15, 1916, the middle finger of his right hand was cut transversely across the palmar surface at the distal interphalangeal joint. The flexor profundus tendon was severed. The wound healed but the distal phalanx of the finger could no longer be actively flexed.

Operation (February 27, 1918). Under local anesthesia. A transverse incision across the distal crease of the finger with a connecting lateral longitudinal incision made a skin flap which was turned to one side to expose the entire palmar surface of the finger. The flexor profundus tendon could not be found. Its proximal end had probably retracted into the palm of the hand, where the patient says he has a point of tenderness on pressure. A strip of fibrous tissue which was adherent to the distal end of the profundus tendon was sutured to the sublimis tendon at the distal end of the proximal phalanx. Wound closed.

Discussion. This operation may prove insufficient to secure active motion of the distal phalanx. If necessary, a second operation can be done to expose both ends of the profundus tendon and to unite them by splicing or by using a portion of a tendon of the foot as a graft.

Case VII (Reported in *Transactions of the Philadelphia Academy of Surgery*, October 1, 1917, *Annals of Surgery*, January, 1918). J. D. Episcopal Hospital, June 18, 1917. The ring finger of his left hand is contractured at the metacarpophalangeal and the proximal interphalangeal joints and is flexed completely into the palm. Extensive dense scar tissue is present in the flexure of the joints. The deformity is the result of an injury sustained twelve years ago. He is a leather-worker.

Operation (June 27, 1917). The scar tissue was excised from the finger, leaving a raw surface extending from beyond the proximal interphalangeal joint to the distal palmar transverse crease. The flexor sublimis and profundus tendons were then lengthened to allow the finger to be extended. It was then found necessary to excise a portion of the distal extremity of the proximal phalanx in order to permit of free motion in the proximal interphalangeal joint. The pouch of skin on the dorsum of this joint was used in part as a flap to cover the flexor surface of the joint. The remaining raw surface was covered by a pedicled skin graft from the left thigh.

Operation (July 11, 1917). The pedicled graft was cut free from the thigh. The graft was firmly united to the hand.

September 17, 1917. The wound of the hand gradually healed after a little sloughing of the graft along one margin. The tendons were exposed for a time but did not slough.

The finger is straight. The skin graft has made a thick pad of tissue at the base of the finger. No active flexion of the finger is present and but slight passive motion. The patient has returned to work.

February 11, 1918. The finger is straight. There is active flexion of the metacarpophalangeal joint to a right angle. No active motion in the interphalangeal joints. Slight passive motion in the distal joint but none in the proximal. The skin graft is thick and prominent. Sensation is present in the graft except along its ulnar margin. This patient can probably be further improved by another operation which will free the tendons from scar tissue, secure better motion in the proximal interphalangeal joint by excision of another portion of the proximal phalanx if it is found necessary during the operation, and which will reduce the thickness of the skin graft.

CASE VIII.—G. W., Episcopal Hospital. In August, 1917, the patient was struck in the forearm by a piece of steel which buried itself in the muscles. At operation the surgeon had difficulty in finding the steel and made a fairly extensive dissection in his search. Finally the steel was withdrawn by the application of a strong magnet. The wound healed promptly without complication.[1]

October 1, 1917. Referred to my service. Examination shows the flexor tendons of the fingers to be adherent to the scar of the incision. The fingers are held in partial flexion and cannot be fully extended either actively or passively. Active flexion is incomplete. There is hyperesthesia and numbness of the middle finger.

Operation (October 17, 1917). Longitudinal incision to one side of the scar. Flexor tendons were dissected free of all scar tissue and were surrounded by a free fat transplant from the thigh. Wound closed.

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[1] Since the publication of this paper the author has learned that following the original operation the wound suppurated.

November 5, 1917. Wound healed, light massage ordered. Good use of hand.

November 19, 1917. Able to make good fist. The fat transplant moves up and down with the tendons. No pain at any time in arm. Normal function of the hand. There is complete active extension of wrist and fingers and almost complete hyperextension of the wrist when the fingers are kept extended. Normal sensation in the middle finger.

Discussion. This case illustrates the fact that a clean operative dissection of the forearm may be followed by adhesions which greatly interfere with normal use of the hand. It is the author's opinion that in any operation, primary or secondary, which requires a dissection of the structures of the forearm, measures should be employed to prevent adhesions of these structures to one another and to the skin. The simplest and probably the best means of prevention is the free fat transplant. Cases IX and X, in which clean operations were also productive of dense scar tissue, confirm this opinion.

CASE IX.—G. F. Episcopal Hospital. In July of 1917 the patient fell from a stack and cut his left forearm transversely to the bones on the palmar side about the junction of the middle and lower thirds. He was treated in a hospital for three weeks, within which time he was operated on.

October 5, 1917. Examination reveals a deep transverse scar on the palmar surface of the left forearm. The arm and hand below the scar are slightly bluish in color and cool, and are without sensation. The patient says the hand "feels dead." There is complete ulnar and median paralysis below the scar, accompanied by wasting of the muscles of the hand. The fingers and wrist are in a position of extension, and there is complete absence of active flexion of the wrist and fingers and thumb except for slight power in the radial flexor of the wrist.

November 19, 1917. The patient has had treatment by massage and has developed very slight active flexion of the metacarpophalangeal and the proximal interphalangeal joints.

Operation (November 21, 1917). Through a longitudinal incision all the structures in the lower half of the forearm were dissected free from dense scar tissue. The dissection had to be carried down to the bones and the interosseous ligament. The ulnar nerve was found fastened to a tendon, the ends of the median nerve were buried in scar tissue, and but one or two tendons were found united. The ulnar artery had evidently been ligated at an earlier operation, as it was not patulous. The dissection of the nerves and tendons was carried down beneath the annular ligament until they were completely free of adhesions. All the tendons were lengthened to permit of their proper suturing. The two nerves also were lengthened and sutured. Fat was then taken from the thigh and placed between the deep tendons and the bones, another layer between the deep tendons and the superficial ones, and a third layer between the superficial tendons and the skin. Tubes of fat were sutured about the two nerves. The skin incision was closed, and the hand and arm were placed on a dorsal splint.

December 24, 1917. Along the line of the old transverse scar the skin sloughed a little, and the underlying fat became infected. The patient has slight active flexion of the wrist and metacarpophalangeal joints, but none in the interphalangeal joints.

January 7, 1918. Some of the superficial tendons of the arm have sloughed out. The wound has been treated from the beginning with dichloramine-T, and it is now healed. There is active motion of the radial and ulnar flexors, slight motion in the metacarpophalangeal joints and some motion in all the joints of the fingers and thumb. Sensation has returned on the dorsum of the hand.

March 4, 1918. There has been steady improvement in all active motions and in sensation.

April 4, 1918. Almost normal active motion of the wrist and about one-half normal active flexion of the fingers and thumb. Sensation has returned to the hand and fingers except in the median and the ulnar distribution. The superficial tendons are adherent to the scar.

Operation (April 17, 1918).—An incision was made at the site of the former one. The superficial tendons were found to be ununited. with their ends separated about two inches, while the deep tendons were all united but were partially adherent to scar tissue. The two nerves were also ununited, and the proximal end of the ulnar nerve could not be found. Again the superficial flexors were sutured after being lengthened. The tendons of the little and ring fingers were sewed together above and below the gap and a part of one tendon was used to bridge the gap. The same procedure was employed with the superficial flexor tendons of the index and middle fingers. The median nerve was spliced and sutured. Fat from the other thigh than the one used at the first operation was placed between the deep and the superficial tendons, and between the superficial tendons and the skin. The hand and arm were dressed on a dorsal splint.

Discussion. A free fat transplant must of necessity secure nourishment and a new blood supply from the surrounding tissues, if it is to live. It may do this with difficulty when the tissues with which it is in contact are scar tissue with but scanty blood supply.

In this case three layers of fat were introduced within the arm, and it is not to be wondered at that the scar tissue in the skin, together with the superficial layer of fat, failed to secure proper nourishment and sloughed. But the surgeon should not be discouraged if in an extensive plastic operation he does not secure an ideal result at the first attempt. If he be able to improve the condition in part by successive operations, a good result will be obtained in the end.

In this case a result from the suturing of the nerves is scarcely hoped for. In fact at the second operation the proximal end of the ulnar nerve was not found. If the tenoplastic work has now been accomplished, the proper nerve-suturing must be attempted at a later operation, when the ulnar nerve may be exposed above the present wound and followed downward.

CASE X.—M. McD. Episcopal Hospital. On August 27, 1913, this patient, a girl of six years of age at the time, fell from an express wagon and suffered a fracture of the right humerus at the elbow. She was taken at once to the receiving ward of the Episcopal Hospital and the arm was dressed in a position of hyperflexion of the elbow. An *x*-ray picture taken the same day showed that the fracture had been reduced. She was dressed again two days later, August 29th, in the surgical dispensary. Blood blisters were present on the forearm. There appeared to be no feeling in the hand and fingers. No pain. On October 7, 1913, she was seen by Doctor Ashhurst who found the following conditions: elbow motion, 75 to 135 degrees. Slough on ulnar surface of forearm two and one-half inches long and one inch wide, which seemed to involve only the skin and subcutaneous tissues. No sensation below the wrist. Beginning Volkmann's contracture. Hand in mid-pronation with scarcely any rotation possible.

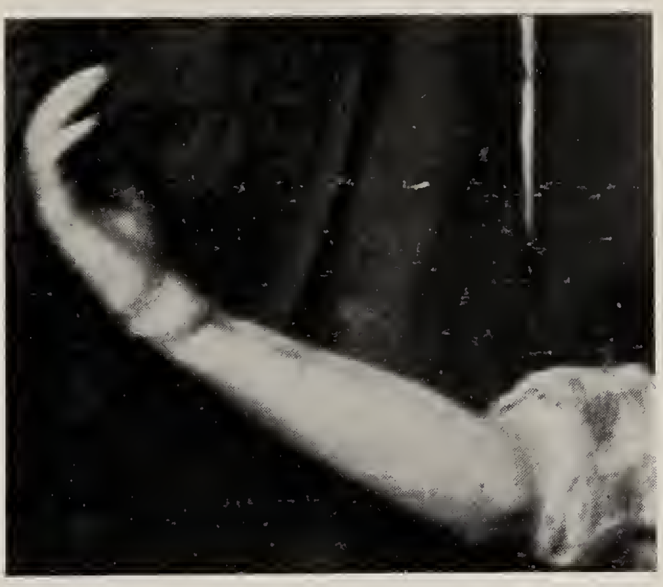

Fig. 12.—Case IX. May, 1918.

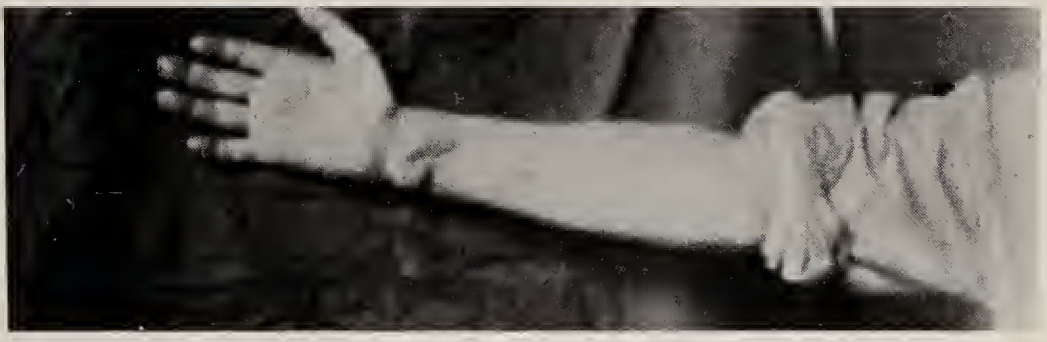

Fig. 13.—Case IX. May, 1918.

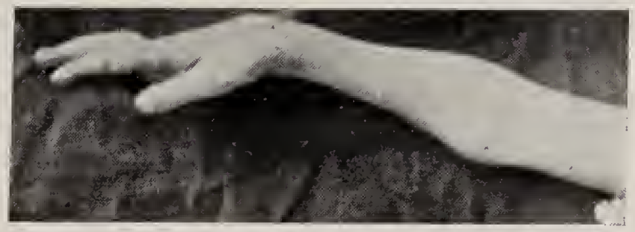

Fig. 14.—Case X. April 9, 1918.

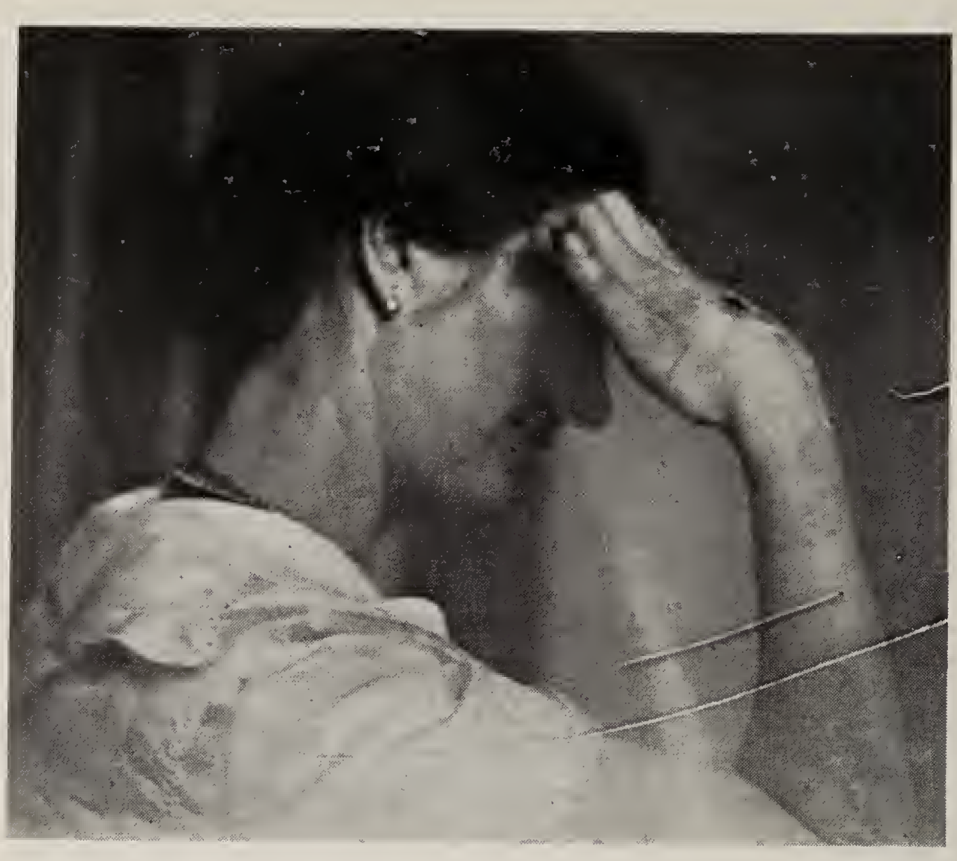

Fig. 15.—Case XI. April, 1918.

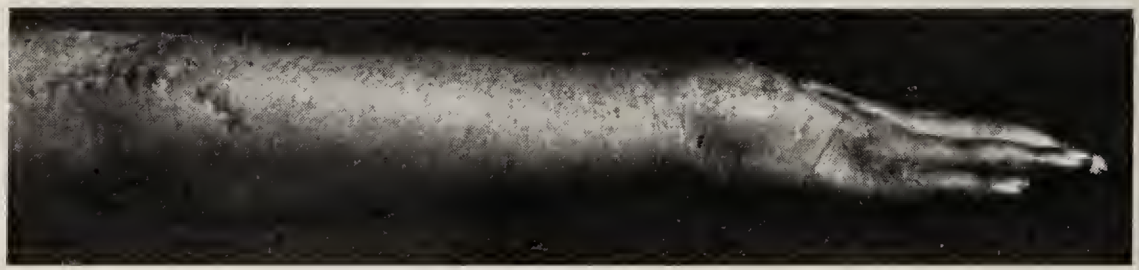

Fig. 16.—Case XI. April, 1918.

November 3, 1913. Motion of elbow 50 to 130 degrees. Granulating wound on ulnar surface 1.0 cm. above the ulnar styloid, 3.0 x 2.0 cm. Wrist extends 20 degrees, flexes 45 degrees. Claw hand. When wrist is flexed to the limit, the fingers can be fully extended. Sensation present in radial nerve distribution on the outer side of the web between the thumb and index finger, and nowhere else. No sensation in median nerve distribution. The only sensation in the ulnar distribution is on the ulnar surface of the fifth metacarpal.

November 24, 1913. Wound healed. Massage and active and passive motion ordered.

December 8, 1913. Sensation has returned in the distribution of the radial nerve. Rotation of the forearm has slightly improved.

December 22, 1913. Slight power in the lumbrical muscles.

January 5, 1914. Fingers can be straightened when wrist is extended. Improving.

February 16, 1914. Anesthesia present chiefly in the ulnar distribution.

March 9, 1914. Wrist can be slightly hyperextended. Dorsal ulnar and median anesthesia, palmar anesthesia distally from web of fingers except the thumb.

Operation (March 25, 1914). By Doctor Ashhurst. Dissection of the structures of the forearm. The incision was closed without placing any fat about the structures beneath.

April 20, 1914. Active movement of the flexor profundus digitorum in the middle and ring fingers; doubtful in index and little fingers. Sublimis active in all. Extensors of fingers slightly active. Thumb flexors and adductors inactive. Massage and passive motion ordered.

October 12, 1914. Elbow motion 30 to 155 degrees. Anesthesia on extensor surface of two terminal phalanges of middle finger and on flexor surface of tips of index and middle fingers. Some power in deep and superficial flexors of the fingers.

August 20, 1915. Thumb much improved. Elbow motion and areas of anesthesia unaltered.

October 1, 1917. The patient disappeared and has had no treatment during the past year. Elbow motion 45 to 155 degrees. The right forearm is much smaller than the left and is one inch shorter. Absence of rotation, forearm in mid-position. The hand is in extreme flexion and cannot be extended to the straight line, and it is deviated to the ulnar side. There is slight active flexion of the fingers, but none of the thumb. The patient does not use the right hand. The forearm is very small and the structures are apparently imbedded in dense scar tissue. There is very little muscle tissue remaining. Operation advised for cosmetic purposes.

Operation (October 7, 1917). By the author. The structures of the forearm were dissected free from dense scar tissue. All the tendons were lengthened to permit full extension of the wrist and fingers. Fat from the thigh was placed between the tendons and beneath the skin. The muscles of the forearm were found to be almost altogether replaced by fibrous tissue.

May 6, 1918. Normal sensation in hand and fingers. Wrist and fingers are in a position of extension to almost a straight line. Normal active flexion of the wrist. Active and passive extension of the wrist to 180 degrees. Slight active flexion of the metacarpophalangeal joint of the index finger, none of the middle finger, and good of the fourth and fifth fingers. Very slight active flexion of the interphalangeal joints of all fingers but the middle one. Complete active and passive extension of the fingers. Abduction, adduction and opposition of the thumb are good, but there is no active flexion. The patient uses her hand for many purposes, and is able to pick up objects between the thumb and index finger.

Discussion. This case indicates that operation for Volkmann's contracture may be useless and even harmful unless layers of fat are placed about the dissected structures to prevent re-adhesion.

This child was operated on to remove the unsightly contracture of the hand. This has been accomplished. But she is developing an unhoped-for function of the hand, and the small wasted bellies of the muscles of the forearm below the elbow seem to be growing larger. There is good power in the extensor group of muscles and good power in the flexors of the wrist. The surgeon should never consider a case hopeless, as some improvement can almost always be secured. Possibly a later operation in this case to free further the flexors of the fingers and the thumb may lead to a development of these muscles which now appear to be wasted away. Tendon transplantation may also improve the function of the fingers and the thumb.

Case XI.—K. B. Orthopædic Hospital. This patient lives in Youngstown, Ohio. In February of 1917 her right arm was injured in a machine. The elbow was broken and the back of the hand was badly lacerated. She was treated in a hospital for a month.

December 22, 1917. An irregular cicatrix is present on the outer aspect of the arm and elbow. The external condyle of the humerus is sharp and prominent. The elbow is fixed in absolute ankylosis at 130 degrees, and the forearm is fixed in almost full pronation. There is but 15 degrees of rotation possible. On the dorsum of the hand is a broad scar which is adherent to the extensor tendons of the fingers. The hand is in a position of slight palmar flexion and ulnar deviation and cannot be extended beyond 180 degrees. Active extension of the fingers is complete. Active and passive flexion of the metacarpophalangeal joints is almost completely absent, and there is but very slight active flexion of the interphalangeal joints.

Operation (January 18, 1918). (1) Excision of the elbow through an external lateral incision. (2) Through a longitudinal incision on the dorsum of the hand the extensor tendons were dissected free from the skin and from the bones of the hand. A free fat transplant from the thigh was placed underneath and over the tendons, and the wound was closed. But very slight passive motion of the metacarpophalangeal joints was possible under ether.

February 7, 1918. Elbow wound healed. Incision on hand has opened slightly at one end. Patient has almost normal motion in interphalangeal joints. She wrote a letter today, the first one for a year.

February 22, 1918. Passive motion of elbow 60 to 160 degrees without pain. Very slight active motion.

March 16, 1918. Normal flexion and extension of interphalangeal joints. Very slight motion in metacarpophalangeal joints. Active motion of elbow 90 to 180 degrees, passive motion 60 to 180 degrees. Lateral stability good. Rotation of forearm is one-half of the normal.

April 4, 1918. Active motion of elbow 60 to 180 degrees, passive motion 35 to 180 degrees without pain. Active rotation of forearm one-half of the normal, passive two-thirds of the normal. Normal motion in fingers except in the metacarpophalangeal joints. A small superficial piece of fat sloughed from the wound in the hand. Wound now almost healed. Use of hand and arm has been showing improvement week by week.

Discussion. In this case also there was slight sloughing of part of the fat transplant, due, probably to the poor vascularization of the skin in the region of the scar. Here no damage was done, however, as the tendons did not become exposed in the wound.

Aside from the subject of this paper there may be noted in this case the good result which follows excision of the elbow for old fractures or fracture-dislocations. This patient now uses her hand and arm for dressing her hair, for feeding herself, for writing and for many other important functions, all of which were lacking before her operation.

These cases illustrate the variety of operative procedures that must be employed: plastic work on tendons, nerves, and skin; skin, fascia, tendon, and fat grafting; excisions and arthroplasties of joints.

Conclusions. Many of the numerous cases of partial or complete loss of function of the fingers, the hand, and the forearm due to infections or to injuries may be greatly improved or entirely cured by proper surgical procedures. It may be necessary to perform a number of operations in the severe cases to obtain a good result. Operative measures should not be postponed too long, particularly in those cases which begin to show trophic changes in the joints. Patient and prolonged after-treatment is frequently required, but in this the patient is usually a willing helper because he notes the continued improvement in his condition. Finally, it may be said that one seldom finds a more grateful man than he who has had restored to him the function of a hand which he had despaired of ever using well again.

THREE CASES ILLUSTRATING END-RESULTS OF CERTAIN METHODS OF BRIDGING DEFECTS IN THE PERIPHERAL NERVES[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

Dr. Ashhurst remarked that he presented these three patients as clinical evidence of nerve regeneration after methods employed to bridge gaps in the peripheral nerves which are condemned by many neurologists and experimental physiologists. Both nerve stretching and nerve-flaps are held to be not only useless but positively harmful; but he believed these cases prove the contrary. No electrical reports of the muscles supplied by the damaged nerves are presented, because he had come to the conclusion after a not inconsiderable experience in such matters extending over a period of seventeen years at the Orthopædic Hospital and Infirmary for Nervous Diseases, that where the voluntary contraction of a certain muscle is visible, such evidence is much more reliable than is that obtained by electrical reaction.

Nerve stretching to bridge gaps he had employed timidly and without much elongation of the nerves in his earlier cases; it was not until after repeated attempts at feeble stretching had showed no permanent damage was done to the nerve, that he had been emboldened to employ such forcible stretching as was adopted in Case III (William B.). And the result in this case is so satisfactory, and the method of operation by lateral anastomosis after stretching the ends until they overlap is so much easier than that by nerve-flaps or free transplants, that he should feel no hesitation in preferring it in future cases. It is no doubt probable that the use of free transplants of fascia lata to surround the anastomosis promoted the return of function; but he believes the chief factor was apposition, without tension, of broad areas of denuded nervous tissue. The idea of union by lateral anastomosis followed recognition of the possibility and desirability of determining the extent of scar tissue in the nerve bulbs not by gradually advancing cross-sections as is usually done, but by a longitudinal splitting of the bulb. During suture the bulbs are still in place and by them the nerve may be easily controlled; and after suture he had not excised them but merely left them as they were. (Cases II and III were present and were examined by Fellows in attendance.)

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[1] Read at a joint meeting of the New York Surgical Society and the Philadelphia Academy of Surgery, held at Philadelphia, February 2, 1920. Reprinted from Annals of Surgery, 1920, ii, 408.

Case I.—*Primary neuroplasty of the ulnar nerve.*

Fred B., aged twenty-eight years, had his left forearm crushed between freight cars early on the morning of December 12, 1909. He was brought to the Episcopal Hospital, in the service of Dr. Frazier, and was operated upon by Dr. Ashhurst about eight hours after the injury. There was a compound comminuted fracture of the radius and ulna, with extensive laceration of the soft parts below the middle of the forearm. The bone ends protruded and the laceration of the skin extended entirely around the forearm, with the exception of about 3 cm. on the extensor surface.

Operation. Ether. Esmarch band below shoulder. After debridement the ulnar artery was found crushed and its two ends widely separated; both were ligated. The ulnar nerve was crushed, nothing but a grease-stained strand of sheath, 3.25 cm. long, joining its ends. After enlarging the incision to within 7.5 cm. of the internal condyle of the humerus, and dissecting the ulnar nerve as high as this, as well as down to the wrist-joint, a gap of 1.25 cm. at least remained between its ends, even when put under considerable tension. After repair and fixation of the fractures, a flap 3 cm. long was turned down from the proximal end of the ulnar nerve and was sutured, without tension, to the distal end by one through-and-through mattress suture and three sutures passing through the epineurium only. All these sutures were of fine silk. The severed muscles were repaired as well as possible, after swabbing the entire wound with very hot 5 per cent. solution of carbolic acid. The limb was dressed with alcohol soaked gauze, was placed on splints and was kept in vertical suspension for twenty-four hours.

The patient's temperature rose to 103° F. on the third day after operation, and there was some suppuration and a great deal of sloughing of skin and muscle; but the wounds were all healed by the end of February, 1910, about two and a half months after the injury. Good union was secured in the radius, but the ulna remained ununited. About a year after operation a sequestrum worked itself out from the radius.

The patient was last examined November 17, 1919, ten years after the operation. He continues his work on the railroad and has no disability from his injured arm. His grip is strong and he has normal power in the forearm, elbow and wrist. The ulna remains ununited. There is slightly diminished sensation in the ulnar distribution to the fourth and fifth fingers but normal sensation in the ulnar distribution to the hand. All motions of the thumb and fingers can be performed: there is absolutely no paralysis of any of the thumb muscles, interossei or lumbricals. He cannot make a perfectly tight fist, but this is due to adhesions of the muscles in the forearm to the cicatrix in the skin. It is to be particularly noted that there is no deformity of the fourth and fifth fingers; there is full extension in the phalangeal joints and full flexion in the metacarpal joints, evidencing regeneration of the ulnar nerve.

CASE II.—*End-result of primary neuroplasty of the median nerve.*

James F., aged fifty-two years, caught his right forearm in a carding machine in the spinning mill where he worked and was at once brought to the Episcopal Hospital. On admission to the receiving ward, October 21, 1916, a large, gaping wound on the flexor surface was found and an Esmarch band was at once applied above the elbow to check the free bleeding. The wound was washed with carbolic acid solution (1 to 40), 1500 units of antitetanic serum was given and he was sent to the operating room.

Operation was done by Dr. Ashhurst two or three hours after injury. There was a lacerated wound extending from the styloid process of the ulna to the internal condyle of the humerus, and on the flexor surface from the radial to the ulnar border, except for about 5 cm. at the upper end, where the wound tapered to a point at the internal condyle. In the lower part of the wound the radius was exposed, with some superficial destruction of the bone surface. The median nerve was exposed and torn, the radial artery severed and the muscle bellies and tendons were exposed throughout, but were so lacerated that their identity could not be determined by inspection.

The wound was cleansed with turpentine, followed by soap and hot water, by alcohol, and finally bichlorid of mercury solution. The proximal end of the radial artery was identified about 10 cm. below the bifurcation of the brachial, and was ligated. The distal end was ligated at the wrist, the intervening portion having been carried away by the injury. The Esmarch band, applied in the receiving ward, was then immediately removed. An area of damaged skin, about 2.5 by 10 cm., was cut away. The ends of the median nerve were next identified and a defect of 5 cm. discovered; even by stretching both ends of the nerve and flexing the wrist it was impossible to make even the damaged ends meet, much less healthy nerve tissue. Therefore a flap was cut from each end of the nerve, the flaps were inverted and sutured end to end, without tension, by fine chromic catgut (Fig. 17). The damaged ends of the nerve were not excised, but two ligatures were tied around each to prevent the flaps from splitting all the way to the ends, as indicated in Fig. 17. The lacerated muscles and tendons were repaired as well as possible and a few silkworm-gut sutures were applied across the wound from one skin edge to the other, but an area about 7.5 by 16 cm. had to be left uncovered. The hand and forearm were wrapped loosely in gauze soaked in alcohol, the limb was placed on a splint and was suspended vertically for the first twenty-four to thirty-six hours, and for the first week the dressings were kept constantly moist by alcohol douches.

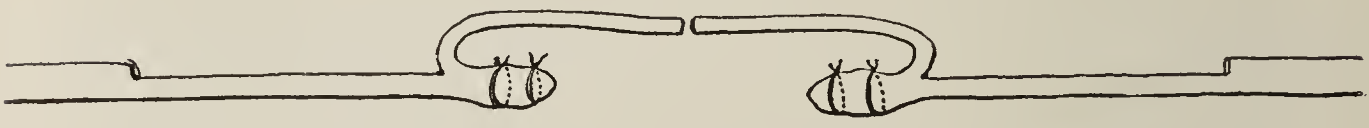

FIG. 17.—Case II. Bridging a gap in a nerve by means of flaps.

Ward Notes. October 23, 1916. Patient claims he has perception of touch in hand and fingers, in area of median nerve distribution.

October 28. Belly of brachioradialis is forming a slough. Pain sensation in ulnar distribution. In middle, index and thumb only sensation of touch. Stitches beginning to cut and removed. Temperature, 99° to 101°. Wound is 17.5 by 8.5 cm. Still alcohol douches.

October 29. Tendon of palmaris longus removed for sloughing.

October 31. Wound clean except over radial side, where there is some exudate. Sensation in thumb, index and middle fingers.

November 1. Temperature normal and steady.

November 28. Sixty-two skin grafts applied by Dr. I. M. Boykin.

November 30. These were all disarranged by the patient moving and today eighty-two others were applied by Dr. Boykin.

December 10. All grafts failed to take (infection).

January 6, 1917. Eighteen grafts by Dr. Boykin.

January 8. Grafts have taken; heliotherapy.

January 31. Discharged. Slight motion in fingers. Is able to flex and extend elbow somewhat. Wrist extends only to 150 degrees or 160 degrees.

February 5. Orthopædic Dispensary, Episcopal Hospital. Skin grafts have taken well, wound healed. Wrist flexed to 135 degrees, cannot be extended. Fingers partly flexed. No active motion except slight flexion of fingers. Hand very stiff. To have massage.

May 21. There is an area of anesthesia of the thumb, forefinger and diminished sensation in the radial side of the middle finger. There is some rotation in the forearm.

October 15. Can almost touch palm with middle and ring finger. Forefinger flexes to right angle. No motion in thumb. Sensation present except on inner side and dorsum of thumb.

October 7, 1918. No active motion in thumb except at metacarpal joint. Can barely touch thumb to little finger; slight passive motion in metacarpophalangeal and interphalangeal joints of thumb. Unable to make a complete fist. Can barely touch the middle and ring fingers to thenar eminence. Little finger about three-eighths of an inch from palm; forefinger about one-half normal flexion. None of the fingers can be completely extended. At work since January, 1918, in spinning mill; not the same work as before injury, but almost same wages.

November 20, 1919. Examined by Dr. Ashhurst.

He was out of work sixty-four weeks in all. He now makes higher wages than before injury, but in a less important position, with the same firm. He uses both hands all day long, in hard manual labor, with grasping actions. There is no anesthesia. All the interossei act; all the lumbricals act. Adduction and abduction of thumb normal.

Can appose thumb to index finger *strongly*.

Can appose thumb to middle finger with fair power.

Can appose thumb to ring finger, mere apposition.

Can appose thumb to little finger with difficulty.

All the voluntary thumb motion is at the carpometacarpal and metacarpophalangeal joints; but if the metacarpal bone is held by the examiner's fingers, slight active motion in the distal phalanx (flexor longus) becomes possible. Impairment of function of the flexor longus pollicis seems attributable to muscular rather than to nervous injury, as the lesion of the median nerve and the site of its suture was below the level at which its branch to the flexor longus pollicis is given off.

He can flex the index finger at the metacarpal joint to an angle of 120 degrees, and at the proximal interphalangeal joint to 90 degrees.

He can flex the third and fourth fingers until they touch the palm, and the fifth finger until it almost touches the palm.

There is slight atrophy on the radial surface of the thumb metacarpal, but no atrophy of the thenar eminence.

He can extend the wrist fully (no hyperextension) and can flex it about to 135 degrees. Elbow motions are normal. Supination of the forearm is slightly limited.

The large scar on the forearm is supple and painless, the identity of many of the skin grafts being preserved; but the underlying muscles are slightly adherent to the skin in the middle third of the forearm and these adhesions somewhat limit motions of the fingers.

CASE III.—*Successful end-result of secondary suture of median and ulnar nerves (lateral anastomosis) and many tendons in the forearm.*

Wm. B., aged eighteen years, on August 28, 1916, broke his fish aquarium and the falling glass cut his left forearm, on the flexor surface above the wrist. An immediate attempt at repair was made in the Receiving Ward of the Episcopal Hospital, by the interne then on duty; a number of severed tendons were sutured and the ulnar artery, completely divided, was ligated. The wound suppurated for several weeks, but healed about October 1. In November, eleven weeks after the injury, the boy was admitted to Dr. Ashhurst's service in the Episcopal Hospital, with a useless hand. There was a cross-shaped scar on the flexor surface of the left wrist, extending to within 3 cm. of the crease of the wrist. The fingers were in full extension and could not be actively flexed, except for very slight power of flexion in the thumb. The fingers were all supple, and could be passively flexed. The wrist motions were normal actively and passively, and the extensor communis digitorum acted normally. There was no action of any of the interossei or lumbricales. There was anesthesia in the median and ulnar distributions below the cicatrices. There was marked atrophy of the thenar eminence and more marked atrophy of the hypothenar. The thumb, as already mentioned, could be flexed very slightly, but could not be adducted, abducted nor opposed. The skin above the wrist was adherent to a dense mass of scar tissue, in which all the flexor tendons were caught.

Evidently this was a case of complete block of the median and ulnar nerves, probably from complete division; also of loss of function of all the flexors of all the fingers and thumb, either from complete division or from inclusion in scar tissue.

Operation was undertaken November 15, 1916, nearly three months after the original injury. Under ether anesthesia, and with Esmarch anemia secured by applying the rubber band just below the shoulder, the longitudinal portion of the old scar was excised and the skin on both sides was reflected. It was densely adherent to the tendinous cicatrix in the middle of the incision, but free proximally and distally.

A. By dissection the following structures were identified: (1) Palmaris longus, its proximal end being lost in the scar; no distal end was found; (2) flexor carpi radialis; by extending the incision through the annular ligament the flexor carpi radialis was traced and found to be carefully sutured to the distal end of the median nerve; (3) proximal end of median nerve, ending in the mass of scar tissue, with a bulbous extremity 1.25 cm. in length. (4) ulnar vessels and nerve, both distal and proximal ends terminating in the mass of scar tissue, and their ends separated by 2 to 2.5 cm.; the ulnar nerve evidently was completely divided by the injury and not sutured; (5) cicatricial mass of superficial flexors, the distal ends of thumb, index and middle tendons not being united to the proximal ends; (6) cicatricial mass of deep flexors nowhere completely divided, but adherent to the underlying bones, and to the scar mass of the superficial tendons. All adhesions were dissected free.

B. The proximal end of the ulnar artery was newly ligated, as the dissection had opened its lumen. The following suturing was done: (1) The flexor carpi radialis was detached from its accurate end-to-end union with the median nerve and was sutured to the flexor pollicis longus; (2) one of the unattached proximal ends of the superficial flexors was sutured to the flexor of the index finger. (3) The palmaris longus was sutured to the flexor of the middle finger; (4) the median nerve ends could not be made to meet even after the dissection, and with the wrist flexed; therefore its proximal end was stretched for 5 cm. by pulling on the bulbous end with hemostatic forceps; in this way the bulb was pulled down past the distal end. Both ends of the nerve were then denuded laterally on apposing surfaces and united by lateral anastomosis by means of three fine silk mattress sutures passing completely through the nerve (Fig. 18); (5) the ulnar nerve was treated similarly, after stretching its proximal end for about 4 cm. In this way broad surfaces of healthy nerve tissue were brought into apposition without tension. The Esmarch band, in place about one hour, was now removed. It was found that no ligatures were required.

C. A portion of fascia lata, about 8 by 10 cm., was next excised from the region of the left great trochanter. This piece was cut in half (4 by 10 cm.) and one of these smaller pieces was again halved, giving two pieces about 2 by 10 cm. in dimensions. One of these small pieces of fascia lata was sutured around the median and the other around the ulnar nerve anastomosis as tubes, being fixed proximally and distally to the nerve sheath by interrupted sutures, the suture for closing the tube in its long axis being continuous. The suture material was No. 000 chromicized catgut, threaded in a fine round needle. The remaining portion of fascia lata, 4 by 10 cm. in dimensions, was arranged so as to form one tube surrounding the bundle of the superficial flexor tendons, including those just sutured; this was to prevent adhesions between the superficial and deep flexors in the depth of the wound and between the superficial flexors and the skin superficially. This portion of fascia lata was sutured around the tendons and fixed by sutures to the fascia surrounding the tendons, as in the case of the nerves. The superficial fascia and the skin were finally closed in separate layers, with

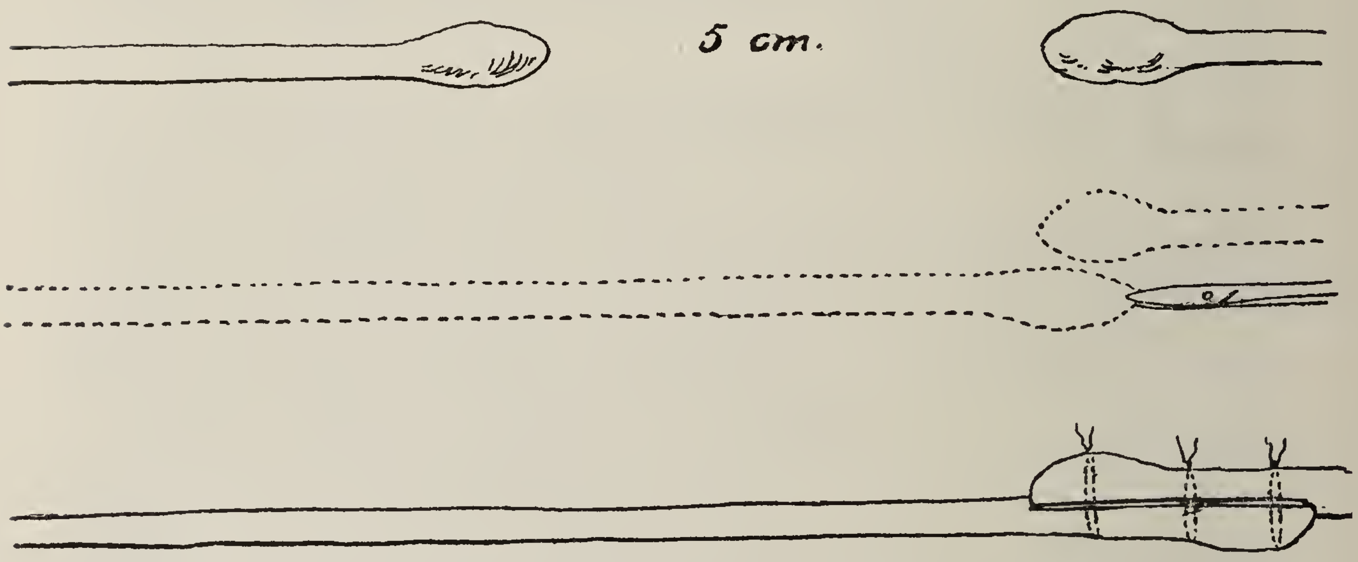

FIG. 18.—Bridging a gap in a nerve by stretching the ends until they overlap. Lateral suture after denudation of apposing surface.

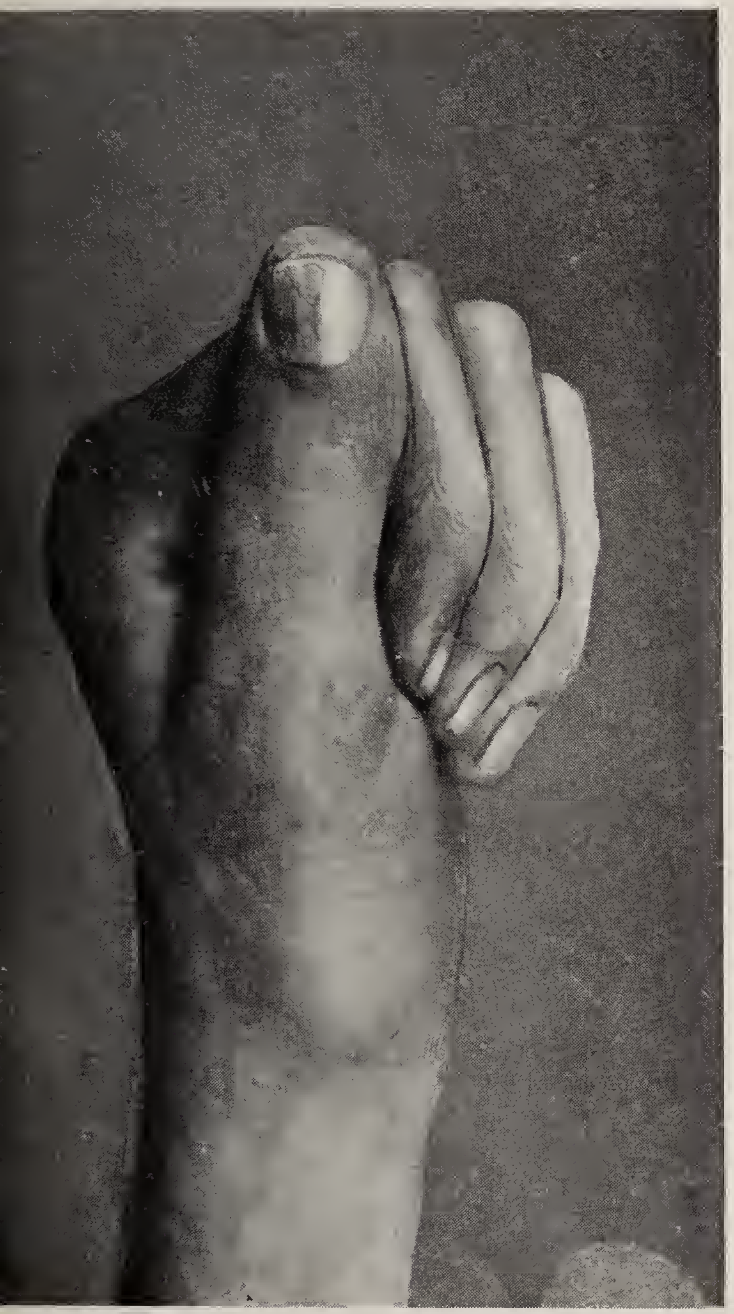

Fig. 19.—Case 3. Three years after sec-
ndary suture of median and ulnar nerves by
nethod shown in Fig. 18. Good grip.

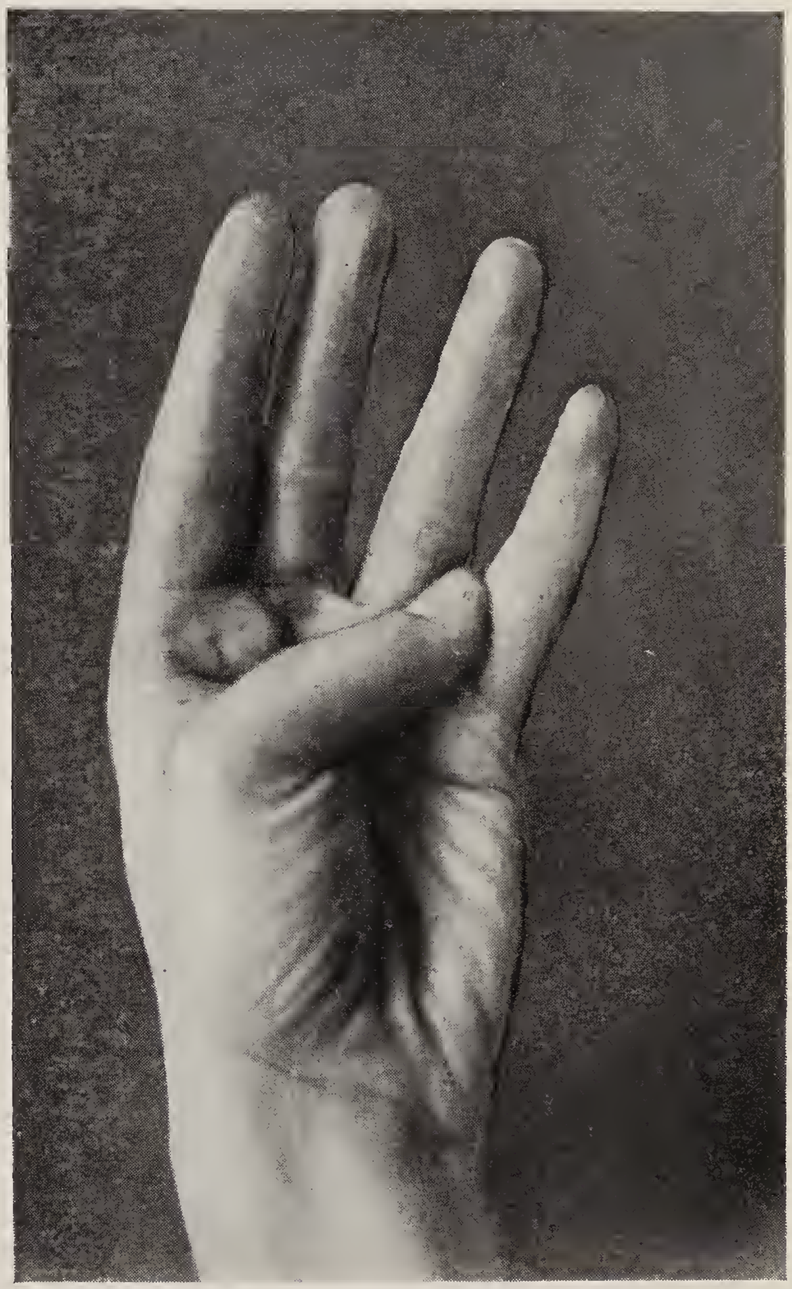

Fig. 20.—Case 3. Three years after suture of median and ulnar nerves, showing opposition of thumb, and action of lumbricals.

many interrupted sutures of No. 0 chromic catgut, and the forearm was dressed on a splint.

The duration of the operation was two hours.

Healing occurred uneventfully. The splint was removed at the end of the second week and treatment by massage was instituted. This was continued for a year after operation, when approximately normal function had been regained. The patient was able to return to his work in a mill three weeks after the operation.

Examination in November, 1919, three years after operation, shows very little trace of the accident. There is slight fixation of the skin to the underlying tendons at one point. Full regeneration has occurred in both median and ulnar nerves, as evidenced by normal action of all the lumbrical muscles, of the abductor, adductor, short flexor and opponens muscles of the thumb, and by very nearly normal action of all the interossei; the only deficiency in the interossei is slight weakness in spreading the fingers apart (Figs. 19 and 20). Each finger can be moved separately in flexion and in full extension, though flexion is possible only until the finger tips touch the base of the palm: they cannot be flexed actively into actual contact with the middle of the palm, but there is normal power in the grip and no disability in hard manual labor. The thenar and hypothenar eminences are less well developed than in the normal hand; indeed, the entire left hand is somewhat smaller than the right. The deep and superficial flexors of all the fingers act separately, showing that they are no longer adherent to each other above the wrist. There is, however, no power in the long flexor of the thumb, its distal phalanx remaining extended when the flexor brevis acts. As the distal end of this tendon was sutured to the proximal end of the flexor carpi radialis, the fact that it does not act cannot be attributed to failure of regeneration in the median nerve, the nerve lesion having been well below the level where its branches to the flexor longus pollicis and flexor carpi radialis are given off. The movements of the wrist, active and passive, are normal.

A REPORT OF TWO CASES OF COMPRESSION FRACTURE OF A LUMBAR VERTEBRA[1]

By THOMAS R. NEILSON, M.D.
SURGEON TO THE HOSPITAL

By a curious coincidence there have come under my observation during my present term of service two cases of compression fracture of the body of a lumbar vertebra.

The first case was transferred to me at the beginning of my term on February 1, 1920, by my colleague, Dr. H. C. Deaver, who preceded me on duty, and to whom I am indebted for the privilege of reporting it.

Case I.—This patient, an Irishman, aged forty-three years, was admitted to the hospital on January 19, 1920, having been injured by falling down the cellar stairs in attempting to escape from his home, which was on fire. The man was brought immediately to the hospital, complaining of pain and tenderness in the lumbar region. While being lifted into bed the pain was aggravated. No definite area of tenderness was noted, the soreness being general, in the lumbar region. The notes state that the extremities were negative. The provisional diagnosis was sprain and contusion of the back.

An *x*-ray taken by Dr. Bromer, on January 20, showed a compression fracture of the body of the third lumbar vertebra. Spinal canal was not encroached upon.

The treatment was rest in bed, with sand bags to effect immobilization.

When I took charge of the case there were no symptoms present, no complaint of pain in the back, bladder and rectal functions normal, as they had been from the first, and the patient's general condition was good.

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[1] Read at a meeting of the Hospital Staff, May 20, 1920; with exhibition of lantern slides by Dr. R. S. Bromer, Roentgenologist to the Hospital.

On March 2, forty-three days after the injury, the patient was discharged in good condition, no symptoms from the vertebral lesion being noted. A day or two before the patient was discharged a plaster jacket, which I thought desirable to prevent for a time motion of the lumbar spine, was applied. The patient was allowed to get out of bed and could walk without any evidence of disability. The plaster jacket was soon found to be very uncomfortable, having shifted its position, and the patient returned to have it removed.

CASE II.—The second case was admitted to my service on April 12, 1920, and is still in the hospital. This patient, a large, well-developed Italian man, aged forty-three years, was injured on the day of admission by a fall down an elevator shaft, a distance of three stories.

On admission his chief complaint was pain in the lumbar region and in the upper part of the left buttock, where there was a slight contusion. Examination revealed tenderness in these regions to palpation; no fracture could be made out. The abdomen was soft, no distention nor tenderness on pressure. The patient could move his legs freely, and there was no apparent limitation of muscular action.

The temporary diagnosis was possible fracture of a lumbar vertebra and of the pelvis (ilium), and an *x*-ray was ordered. The treatment ordered was simply rest in bed on the back.

The *x*-ray plates made on the day of admission by Dr. Bromer showed a compression fracture of the second lumbar vertebra, with no encroachment upon the spinal canal.

Between April 14 and May 14, when I was temporarily not on active duty, I made no examination of the patient, whose condition had remained good in the meantime. There had been no loss or interference with function of bladder or rectum, and the patient had complained of no discomfort, save for that due to an incidental furuncle on the right buttock.

On May 14 the man complained of some numbness of the left leg and foot and that he could not move the toes of that foot as well as those of the right. On examination I found that the loss of sensation complained of was very questionable. Careful testing with one and two pins at various places in both legs and feet failed to reveal any very notable difference, nor was there any, but perhaps the least difference in the ability to flex and extend the ankles and toes of both sides, the motions on the left side being *possibly* slightly restricted. These tests were made with some difficulty because of the patient's inability to understand questions or to make movements as directed, owing to his inability to easily understand English.

On May 18 and 19, I repeated the tests of sensation and motion of legs and feet with similar results, and extended the examination to include the reflexes. The knee-jerks are apparently diminished, but it was impossible to get the man to wholly relax the thigh muscles while the knee was held up during the test. The cremaster reflexes are present but less active than normal. There is no ankle clonus on either side. Plantar reflex present and equal. The patient can easily elevate from the bed the extended limbs, can rotate them equally well and can cross one over the other, the right perhaps slightly better than the left. This may be due to a newly complained of tender area on the anterior surface of the left thigh in the region of the quadriceps tendon, apparently deep seated, but without, however, any palpable lesions to account for it. An *x*-ray of this portion of the thigh will be taken. Measurement of the limbs for length was absolutely equal.

Owing to the patient's tendency to indulge himself by turning from his back in bed, large sandbags are placed beside his trunk and lower extremities to restrain him. He will be kept in bed for some time yet, his symptoms watched, and before he is allowed to get up a plaster jacket will be applied.

In order to clear up any doubt as to the cause of the apparent diminution in the reflexes a Wassermann test has been ordered.

In conclusion, I would briefly call attention to the absence of striking symptoms in both of the cases to direct the attention to the actual lesions, owing, of course, to the fact that in neither was there encroachment of bone fragments upon the spinal canal to cause pressure upon the cauda. Without the *x*-ray both would easily have passed as contusions or sprain of the back.

It is possible that in the second case there may have been some slight stretching of the cord, or a minute hemorrhage, resulting in the very slight disturbance of sensation in the left foot and leg and the diminution in the reflexes.

Just exactly how these compression fractures occurred in these patients is not clear from the histories. It is probable, however, that in both cases the men in falling landed upon the sacral region, which became a fixed point, and that the vertebral compression resulted from sudden, forcible flexion of the spine above the lumbar region. The absence of injury of the lower extremities in either case would evidently exclude the possibility of the fracture being due to vertical pressure from landing upon the feet.

SCREW FIXATION IN JOINT FRACTURES[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

As a general rule, accurate reduction (anatomical reposition) of the fragments in a fracture near to or involving a joint may be secured by bloodless manipulation. It is more important to secure anatomical reposition in such fractures than in fractures of the shafts of long bones; but it is also easier. In joint fractures failure of accurate reduction entails recovery not merely with deformity but also with marked limitation of joint motion; yet fortunately as one fragment is very short the position of this fragment usually may be controlled by the position in which the neighboring joint is dressed. Thus in supracondylar fractures of the humerus, hyperflexion of the elbow secures anatomical reposition of the lower fragment in contact with the diaphysis. In fractures through the neck of the femur, abduction of the thigh restores the fragments to their normal relations. In fractures of the eversion type at the ankle, proper inversion of the foot reduces the deformity. So likewise with the shoulder, the wrist and the knee, modification of the position in which the limb is found, often—indeed I may say usually—suffices to secure proper reduction of the fragments. But every once in a while, a case of fracture near a joint will be encountered in which for one reason or another bloodless manipulations, even if several times repeated under anesthesia, will prove unsuccessful in obtaining reduction.

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[1] Reprinted from Medicine and Surgery, October, 1917.

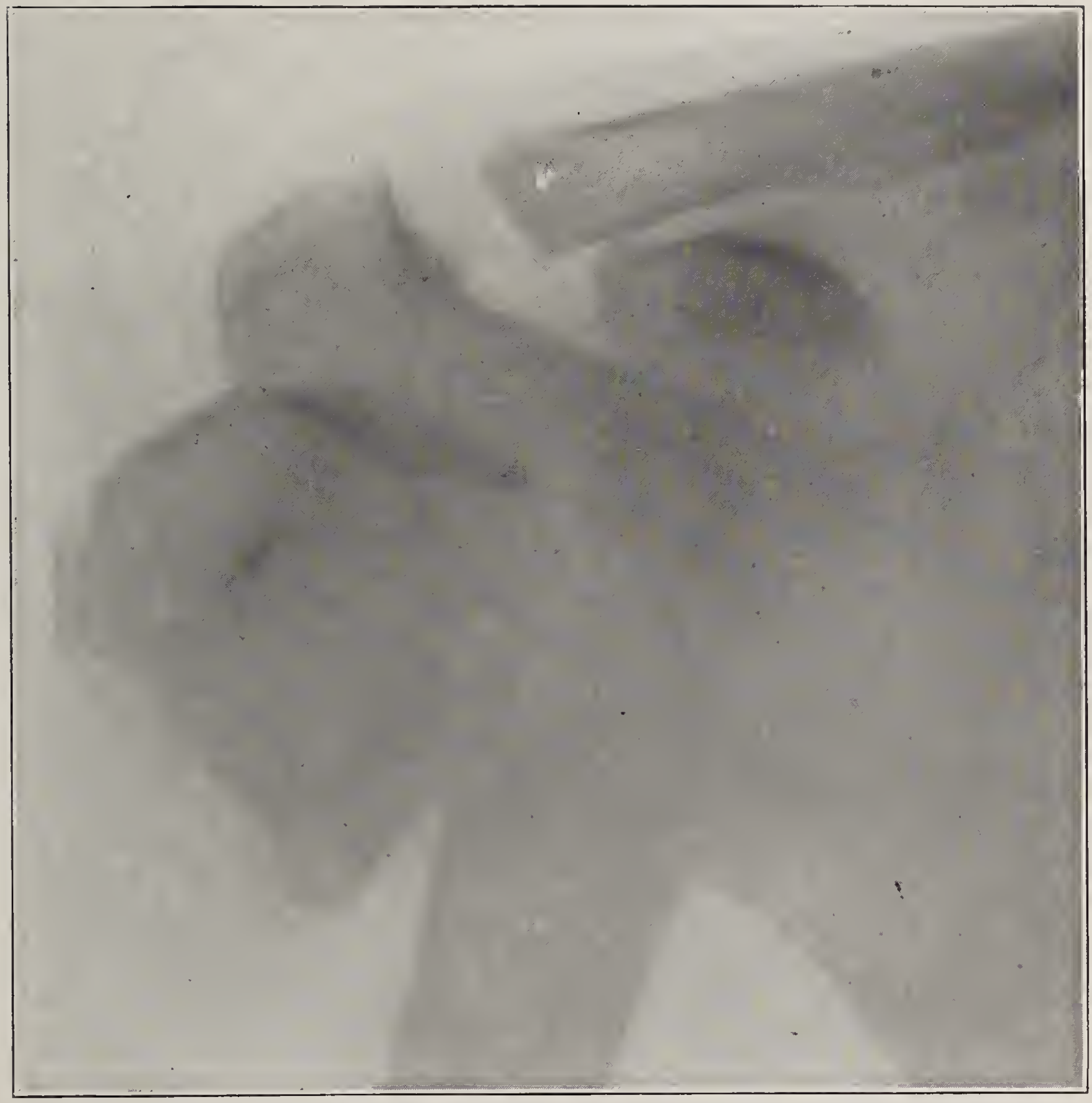

FIG. 21.—Man, aged sixty-two years. Fracture of surgical neck of humerus irreducible even under anesthesia. Also fracture of radius (Colles) of same arm.

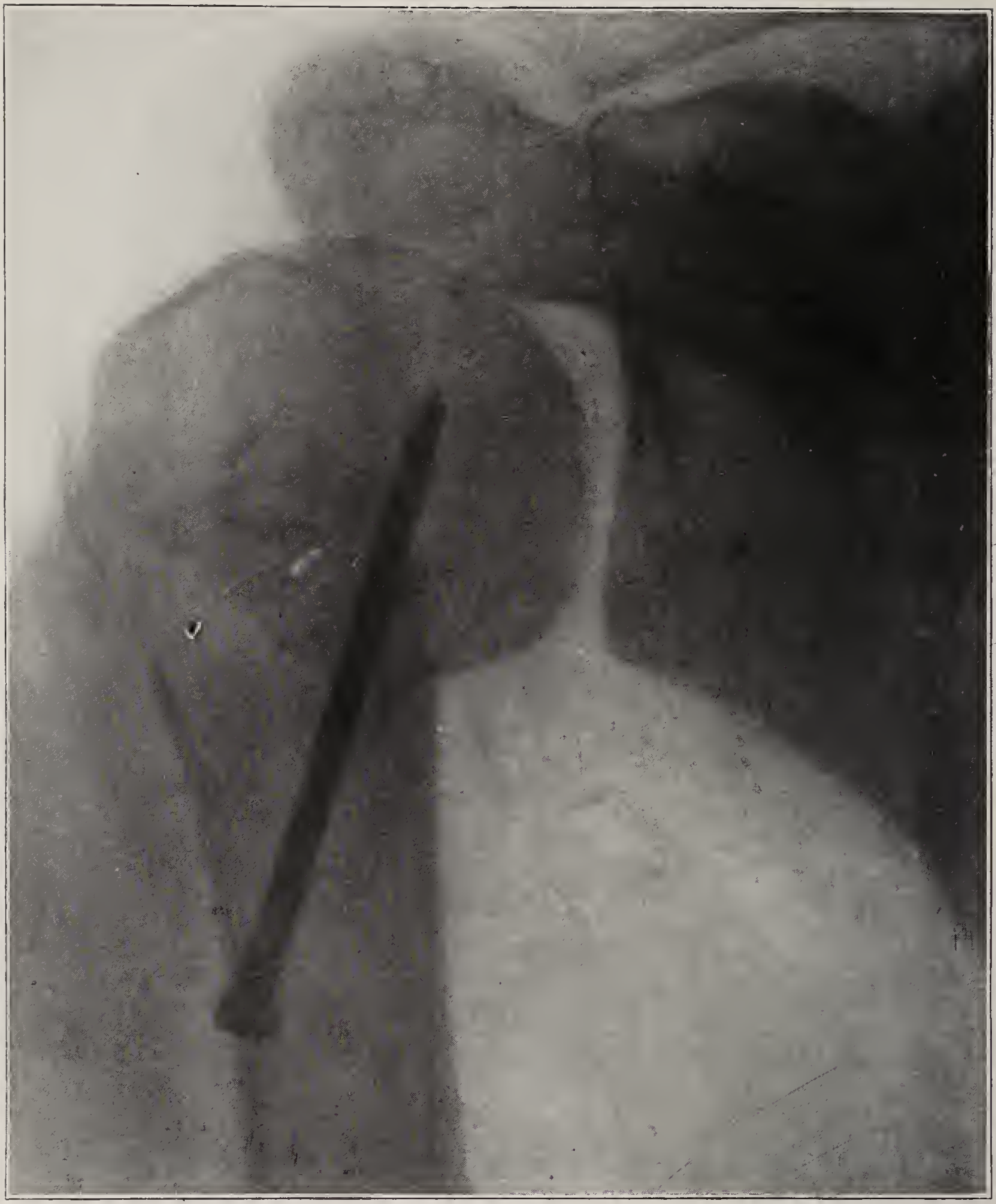

FIG. 22.—Operation seventeen days after injury. In hospital twenty-three days Out of work three months in all. Recovered with same earning capacity, but slightly less endurance.

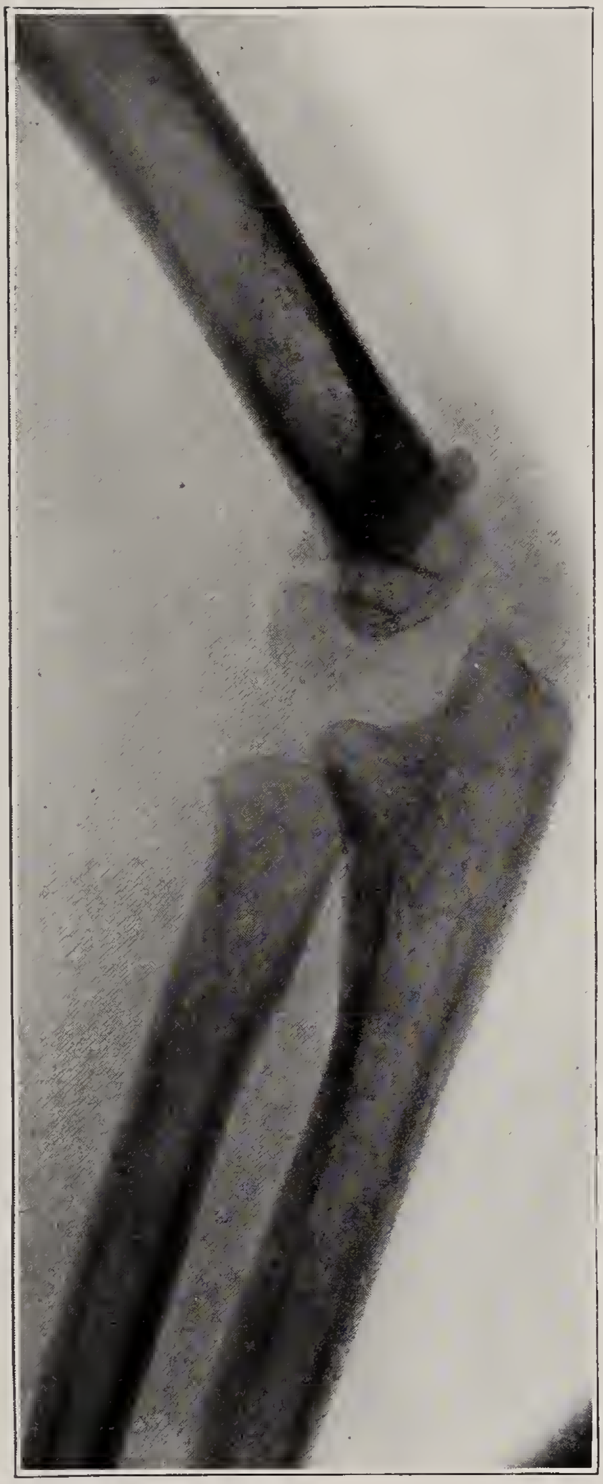

FIG. 23.—Boy, aged eight years, with non-union of fracture of external condyle of humerus, duration five months.

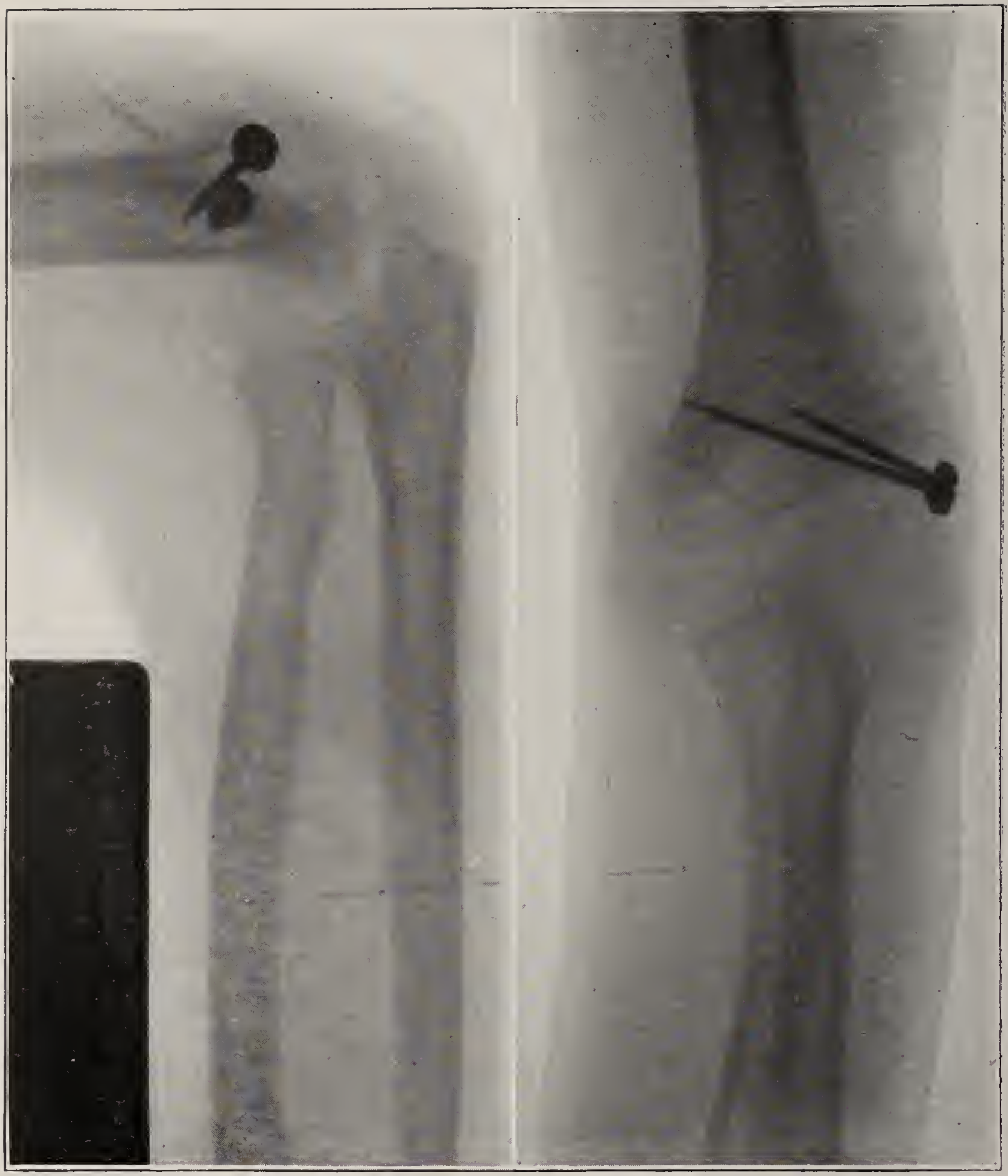

FIG. 24 FIG. 25

FIGS. 24 and 25.—Skiagraphs one month after screw fixation of external condyle of humerus.

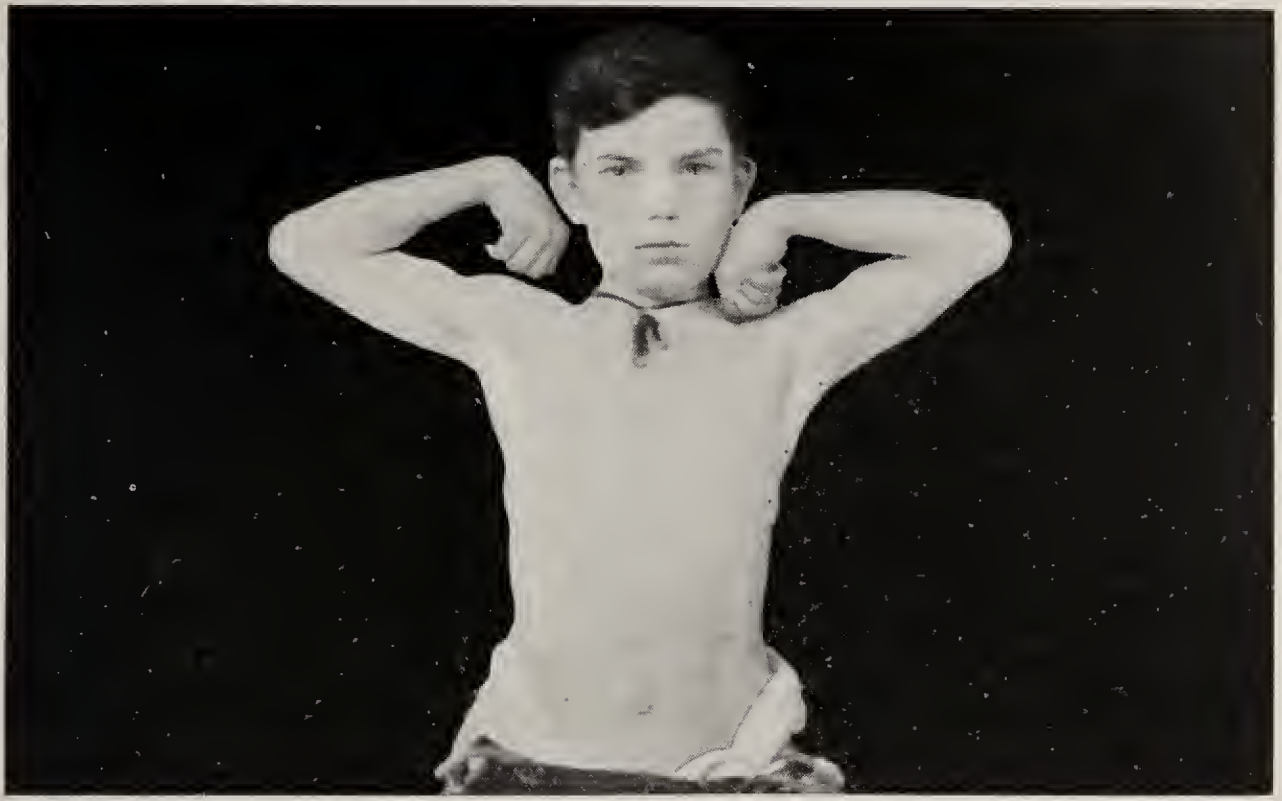

FIG. 26.—One year after operation for non-union of external condyle of right humerus.

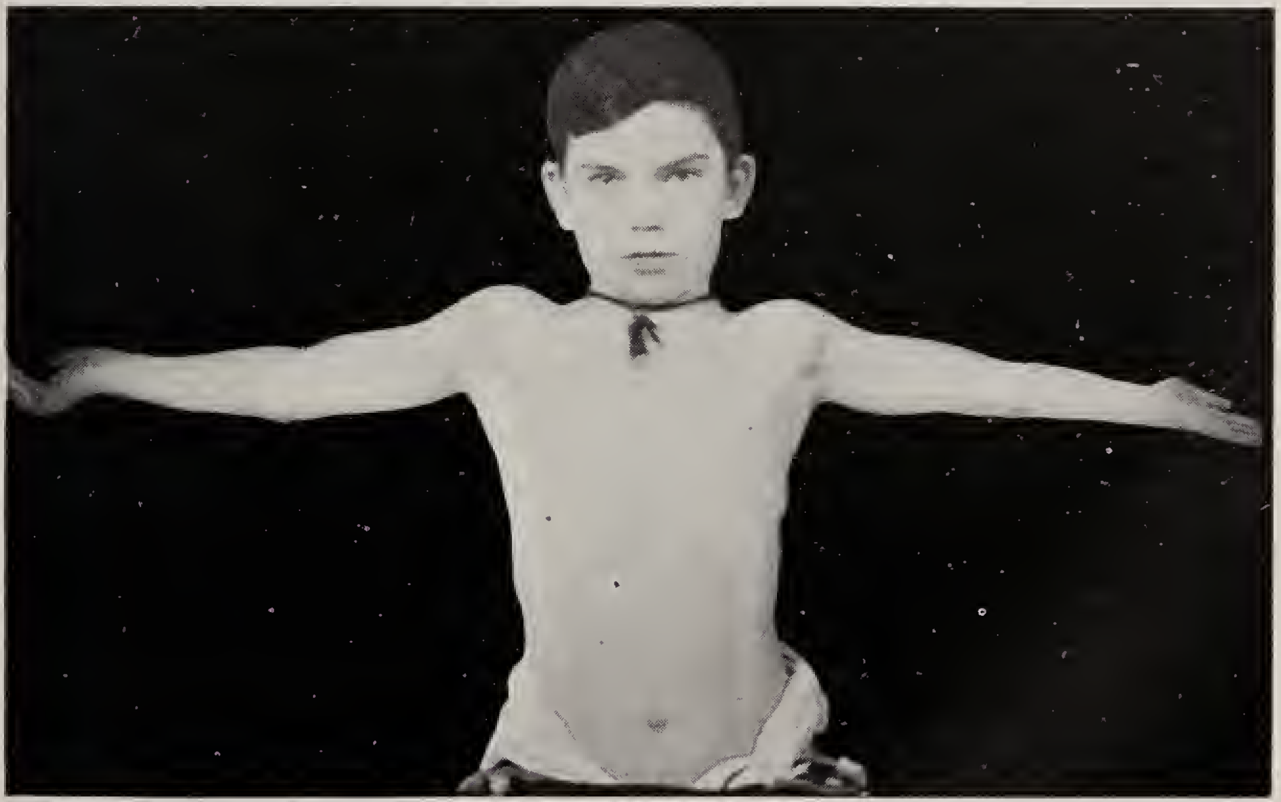

FIG. 27.—One year after screw fixation of external condyle of right humerus for non-union, with displacement, of five months' duration.

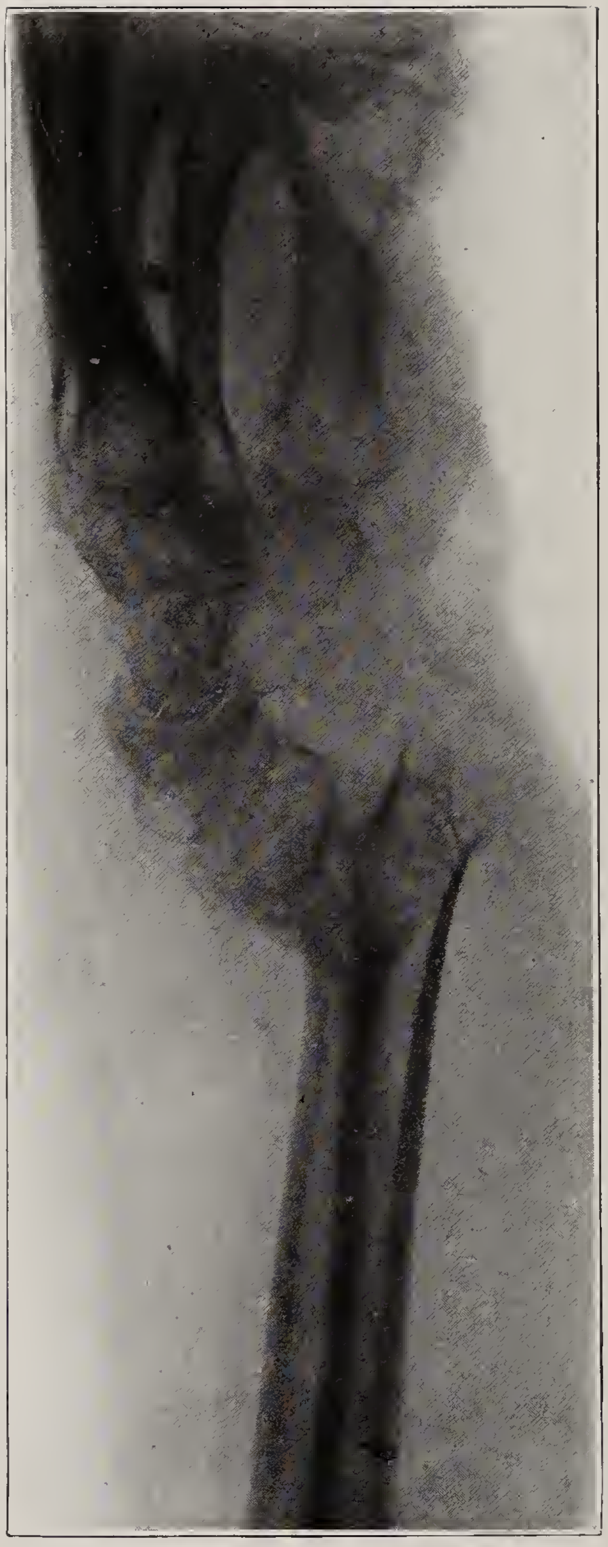

FIG. 28.—Woman, aged twenty-eight years, with unreduced Colles's fracture of four months' duration. Very marked deformity, with involvement of median nerve in callus. Unable to do her work (mill hand).

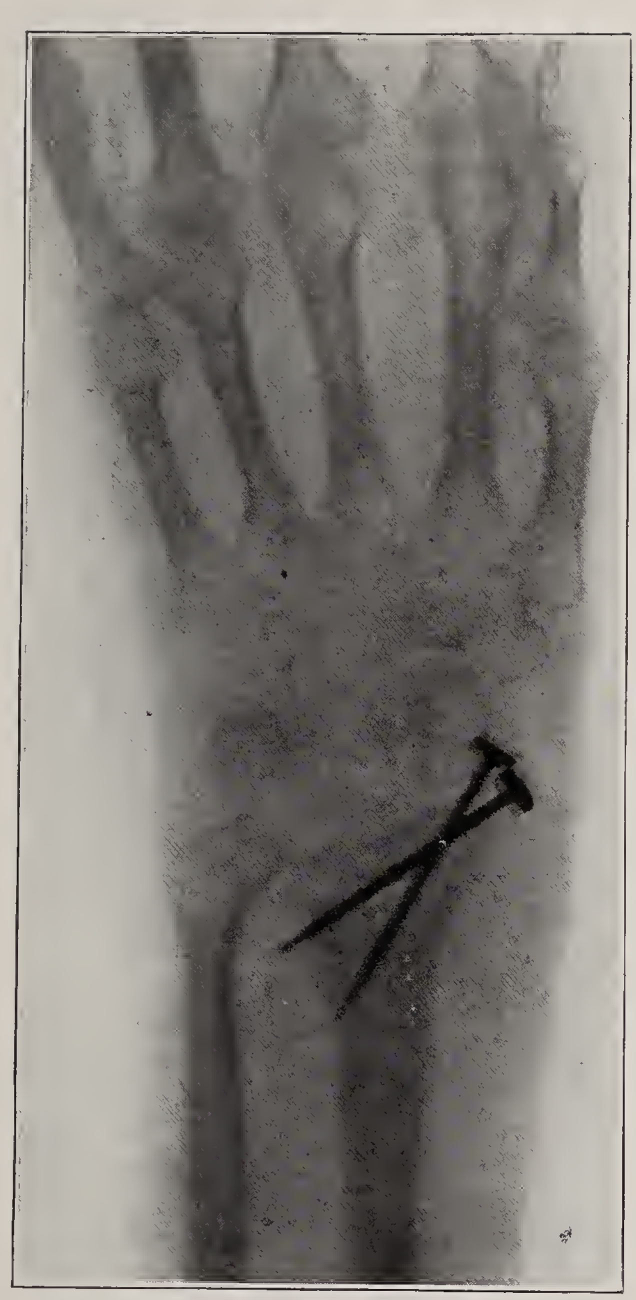

Fig. 29.—One month after reduction and screw fixation. Resumed usual work eight weeks after operation.

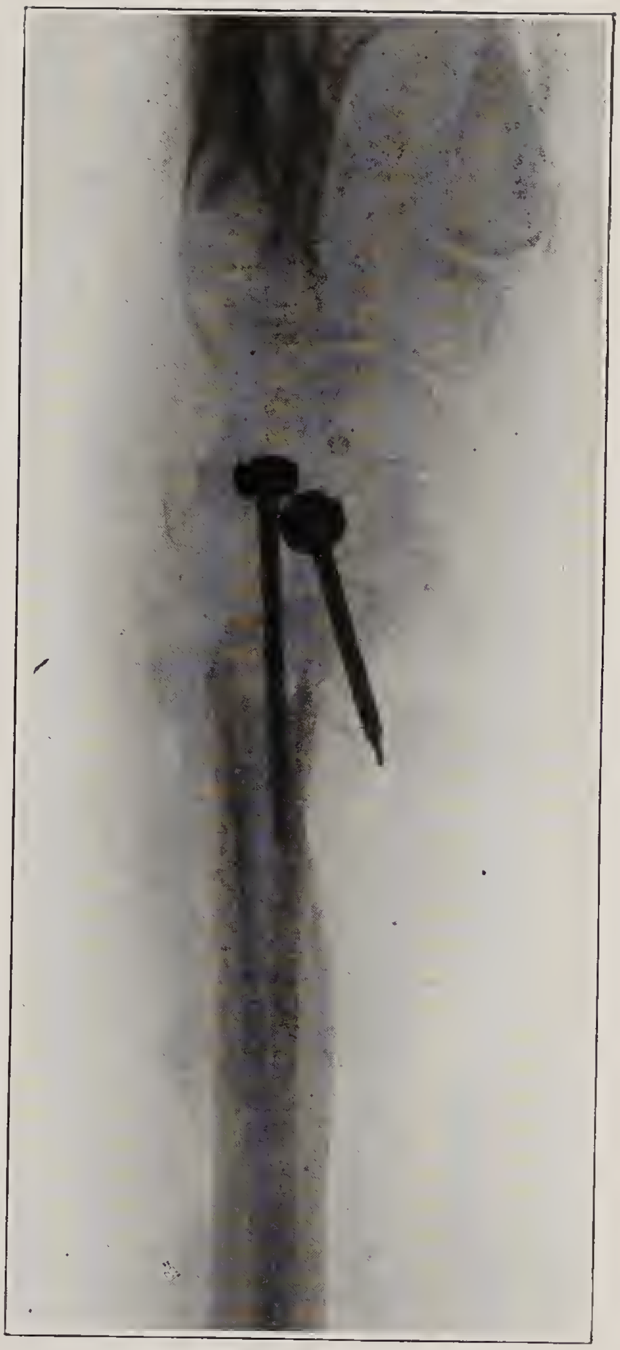

Fig. 30.—Lateral view of Colles's fracture, one month after operation.

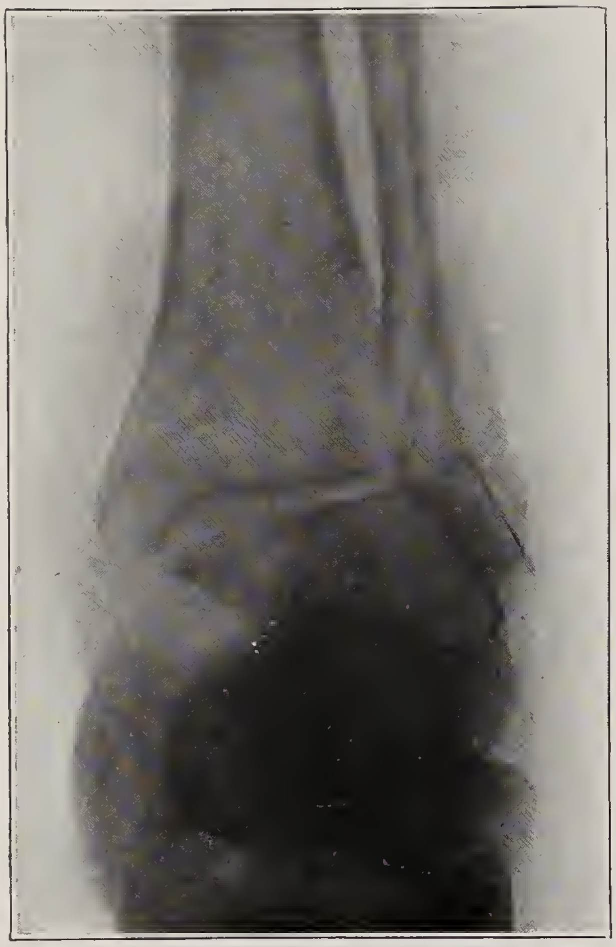

FIG. 31.—Woman, aged fifty-six years, with fracture of both malleoli and extension-eversion fracture at ankle. Failure of accurate reduction would have entailed persistent disability.

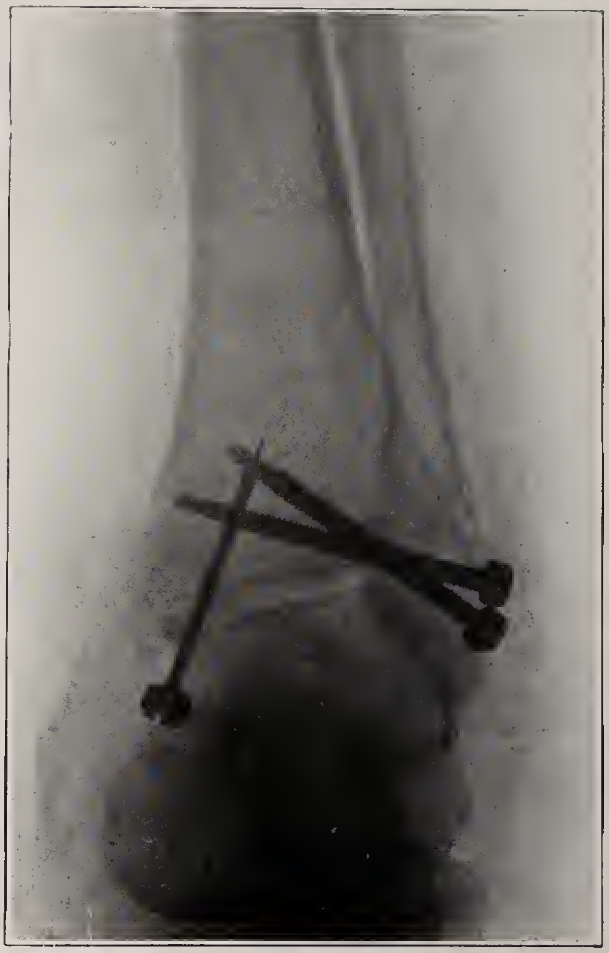

FIG. 32.—Absolute anatomical reposition secured by open operation and maintained by screw fixation.

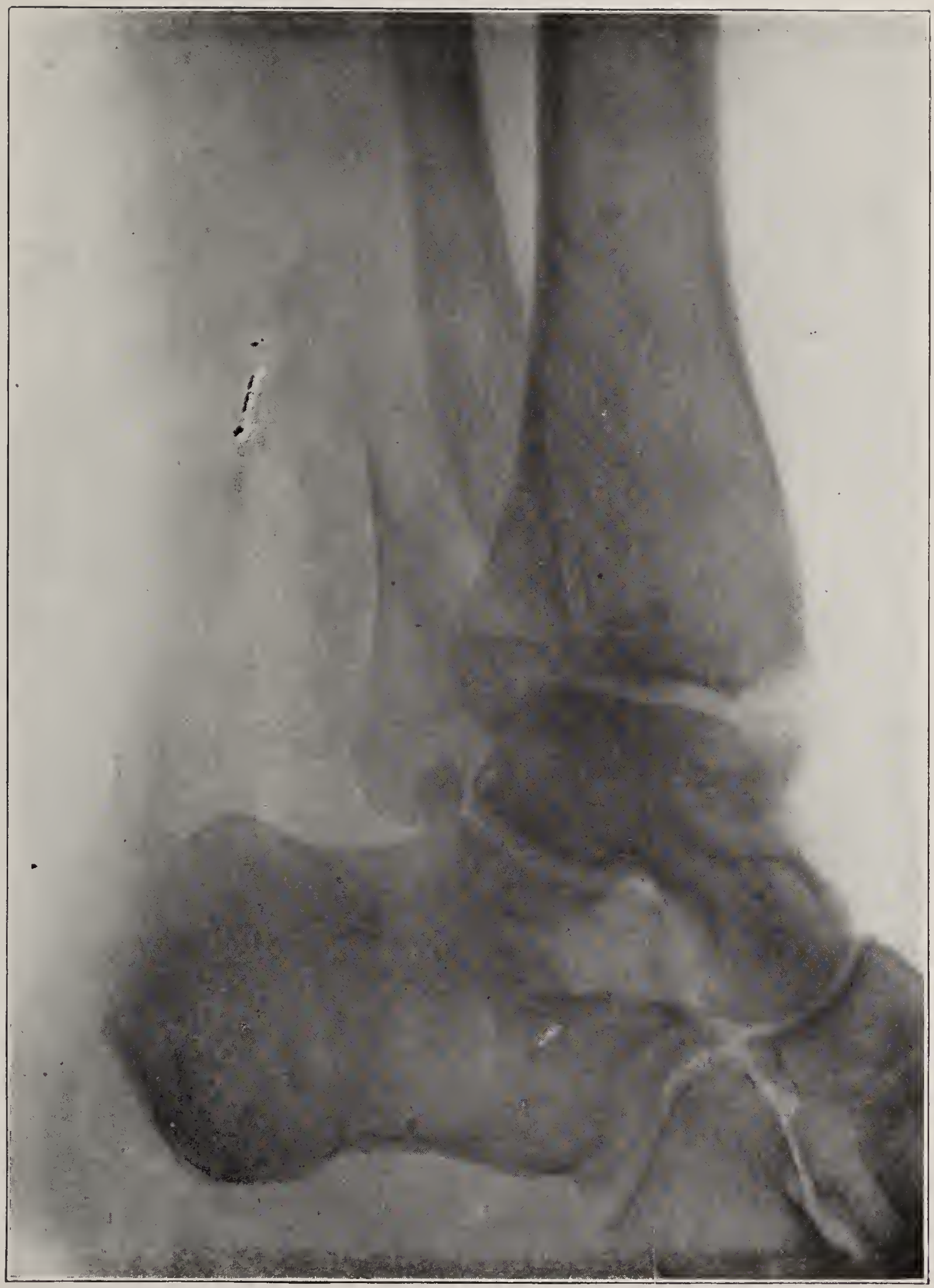

FIG. 33.—Lateral view of fracture shown in Fig. 31. Note the calcareous posterior tibial artery.

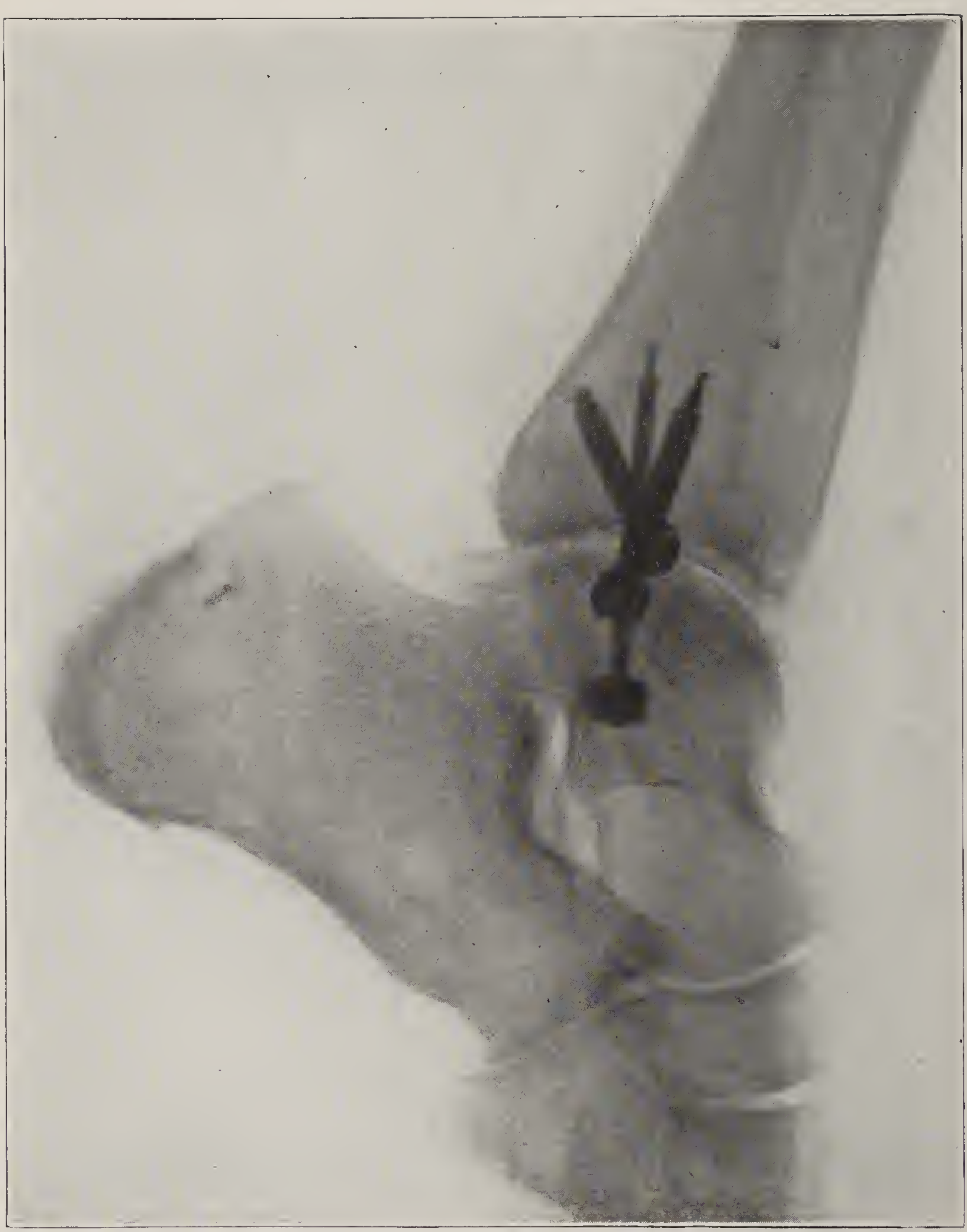

FIG. 34.—Lateral view of ankle after screw fixation for extension-eversion fracture.

In such exceptional cases reduction should be secured by open operation, and it is usually advisable to fix the fragments in proper position by buried prosthesis. This is so for two reasons: if the soft parts have been widely separated from the bones by the original injury, or if the entanglement of the fragments has made necessary such wide separation at the time of operation in order to secure reduction, it is seldom possible to obtain secure locking of the fragments by the position of the limb alone; in the second place, the use of buried prosthesis of some variety has the inestimable advantage that when once the soft parts have healed (by the end of ten days or two weeks) it is possible to treat the limb in many cases much as if the fracture had already firmly united, since the buried prosthesis maintains reduction of the fragments even when the limb is actively moved, provided, of course, the range of motion is not too great.

In this country, plates have usually been used for fixation of joint fractures, but they have the disadvantage of securing a very poor hold upon the short fragment, as well as being sometimes rather difficult to apply. Nail fixation has also been used in many reported cases, but here the objection is to the possibility of splitting the fragments and thus complicating rather than simplifying the cure; moreover, the fixation is not very secure. Ordinary wood screws have also been used, but not to any great extent; and to them the objection applies that the procedure is unnecessarily complicated, inasmuch as it is first necessary to drill the holes and then insert the screws. The latter objection (that of the necessity of first drilling the holes) applies also to the use of ivory or decalcified bone pegs and to pegs made from the patient's own bone. Valuable as auto-transplants are in cases of non-union, they are unnecessary in recent fractures and in most cases of old fracture with malunion; and since a much simpler and more efficient method of fixation is available, I believe their use in such cases should be discouraged.

The method of screw fixation I have employed for the past several years is that of Lambotte, of Antwerp. No one who has used Lambotte's screws, I venture to assert, will fail to be pleased with the technic of the operation and with its results. These screws were devised by Lambotte for use only in the cancellous tissue at the end of bones; they are *self-boring* screws, but are not intended to be able to bore their own way through compact bone, and should not be put unnecessarily to such a test. The thread on these screws has less inclination than the thread of the ordinary wood screw, but more than on machine screws.

It is necessary merely to reduce the fracture, to clamp the fragments securely with the bone forceps, to insert the self-boring screw or screws, to remove the forceps and close the soft parts. The great advantage is that once the fragments are reduced, the first screw inserted holds them in position; there is no possibility that after a hole is drilled for the screw or the peg, the fragments may slip and that thus when the surgeon attempts to insert the fixation apparatus he may find the drill holes no longer in alignment.

The self-boring screws have flat heads, of such shape that they fit into a special holder which is adjustable to any ordinary brace. The screw point is then placed against the bone, the drill rotated until the screw is firmly fixed in the bone, the brace with attached holder is then removed from the screw, which is finally screwed all the way home by an ordinary screw-driver. In most cases it is well to employ two screws in order to prevent rotation of one fragment on the other; and the screws should not be placed parallel to each other—even at a slight angle one screw binds the other, and makes separation of the fragments almost impossible. It is not necessary to countersink the screw heads further than is done by screwing them down upon the cortex as tight as possible; usually the periosteum or joint ligaments rise to a level with the screw heads. But it is advisable to avoid placing the screws in a subcutaneous position exposed to trauma, as for instance, directly over the epicondyle or epitrochlea at the elbow or over the subcutaneous surface of the external malleolus.

I have employed these screws in 26 patients (54 screws in all), and have not had to remove any of them on account of irritation. In one case of operation for malunion of an ankle fracture the screw was removed about a year after operation because the pain present before the operation was not entirely relieved, and it was thought possible the screw might be the cause of the persistent pain; but as its removal made no change in the symptoms, it is evident it was not the cause. In another case of operation for old fracture of the elbow, the end of the screw was palpable under the skin over the epicondyle, and caused annoyance to the patient, but no inflammatory disturbance; it was subsequently removed at a second operation undertaken in an effort to secure a greater range of movement.

There are appended a few skiagraphs of fractures in which screw fixation was employed.

FRACTURES OF THE CARPUS; WITH A REPORT OF 15 CASES: 12 OF THE SCAPHOID, 2 OF THE SEMILUNAR, AND 1 OF THE PISIFORM

By WILLIAM WHITAKER, M.D.
SURGEON TO THE DISPENSARY OF THE HOSPITAL

It seems worth while to call attention to the frequency with which fracture of one or other of the carpal bones is met with in the surgical dispensary.

Those fractures resulting from crushing wounds or from puncture wounds are not included in this series of cases. The majority of these cases have been the result of falls upon the outstretched hand, and of the fifteen cases noted here, fracture of the pisiform bone has occurred once, fracture of the scaphoid twelve times, and fracture of the semilunar twice.

The failure to recognize early a fracture of the carpal bone has resulted in the unsatisfactory results often obtained and has been the cause of a great deal of discomfort and disability to the patient. The broken scaphoid has little power of repair and seems to have but slight power of callus formation. An ununited fracture of this bone is capable of producing symptoms for months or years, and if left untreated is likely to be the cause of an arthritis, sufficient to interfere with a satisfactory result being obtained even though operation and excision of one or more fragments is finally resorted to.

The common mistake made in these cases is to have them diagnosed as sprain of the wrist and thus to have lost the first valuable weeks when fixation is of the most avail and when by proper diagnosis and treatment there is possibility of getting

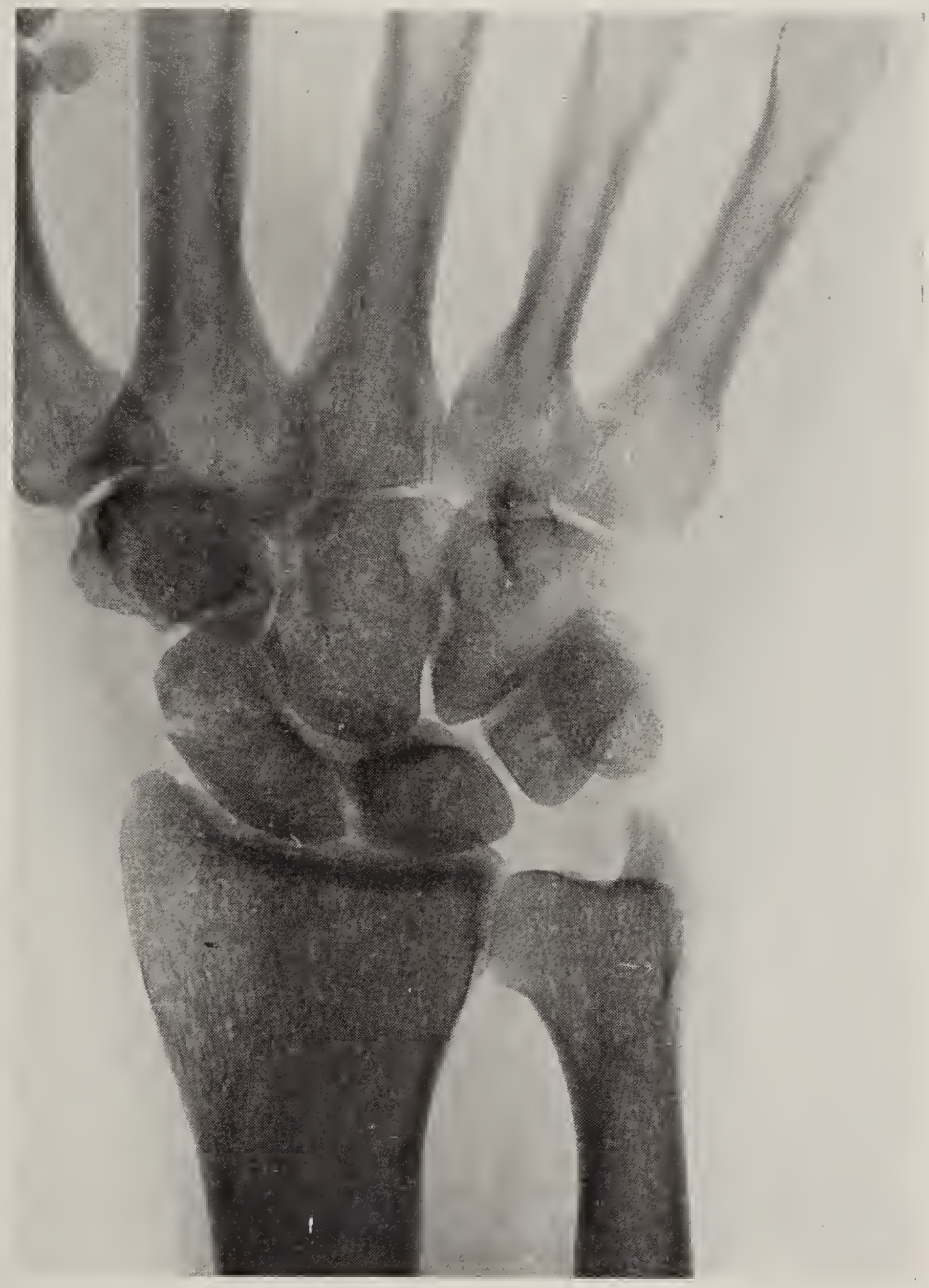

FIG. 35.—Case I. Fracture of the carpal scaphoid.

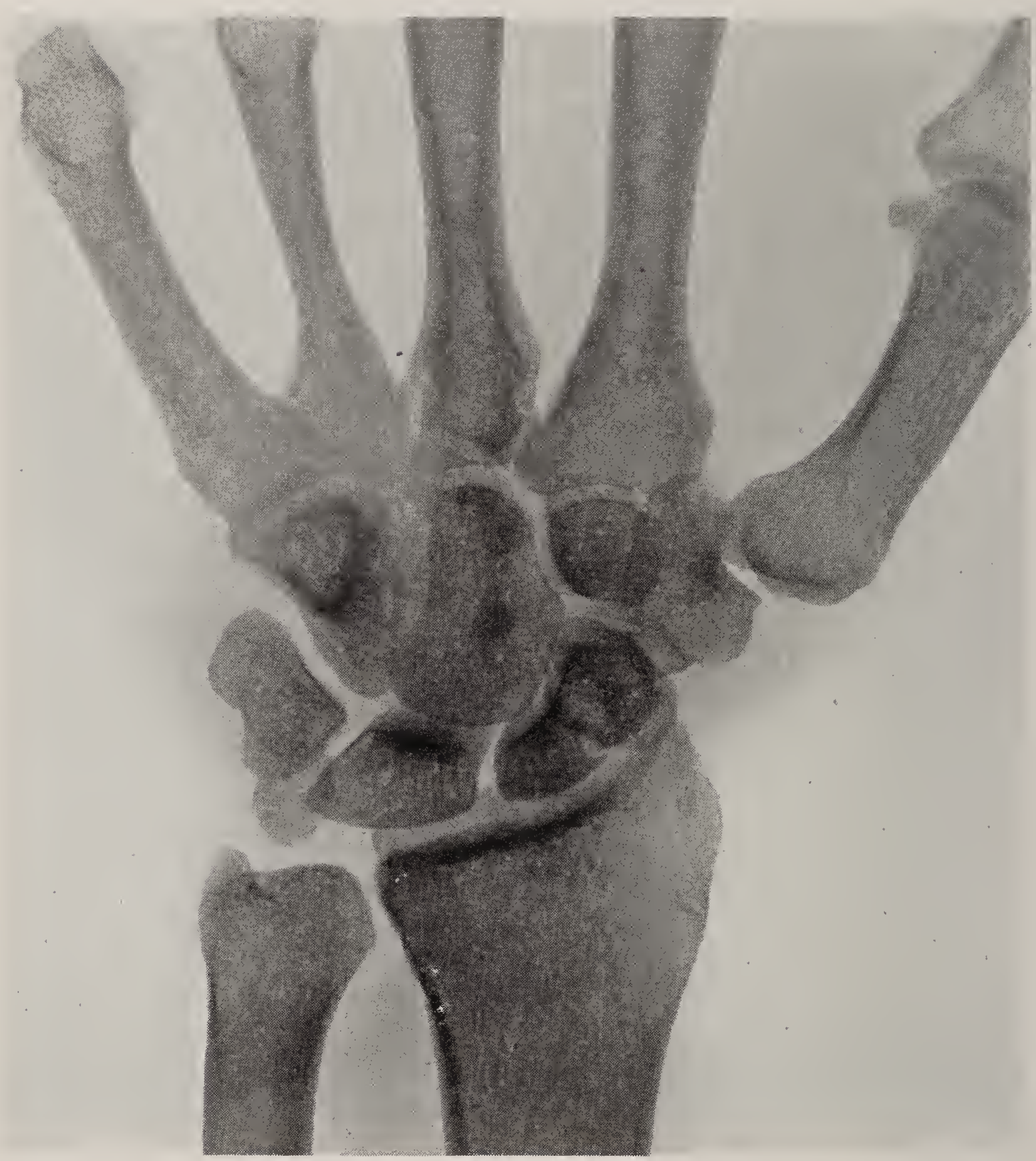

FIG. 36.—Case 3. Fracture of the carpal scaphoid.

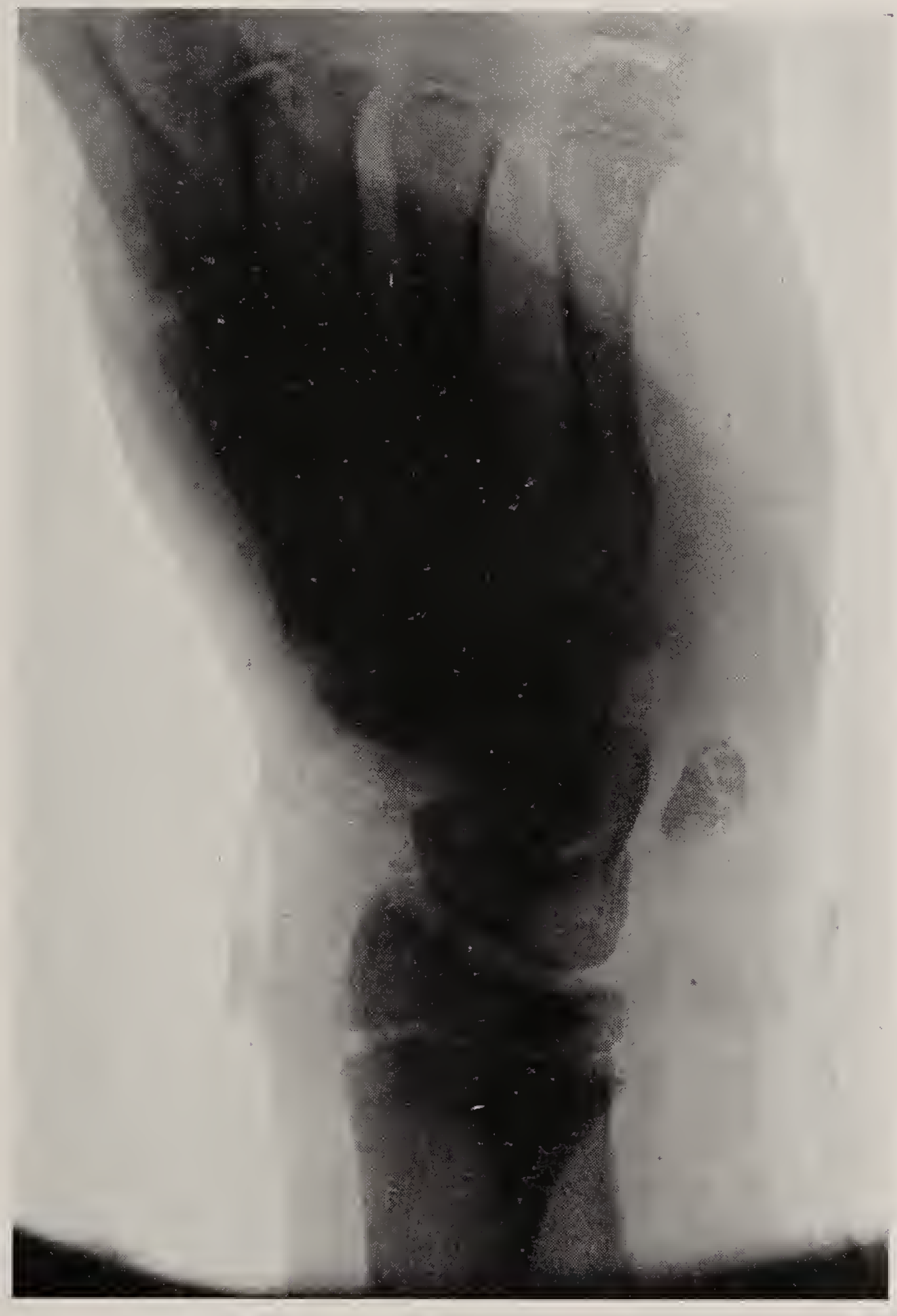

FIG. 37.—Case 15. Fracture of pisiform bone.

bony union between the fragments. Too frequently it is only after the symptoms suggesting a sprain have persisted for an undue time that finally an *x*-ray examination is made and the fracture is found. Unless these cases are diagnosed and fixation established in the first three weeks it is not at all likely that bony union will occur.

Unless an *x*-ray examination is made an early diagnosis is always surrounded by obscurity. In none of the cases reported here is there any record of ecchymosis or crepitus being found. There has been usually only a slight amount of swelling. Pain in the wrist, especially on active and passive motion, has been a constant symptom, and the arc of extension of the wrist is reduced perhaps as much as one-half. Examination has disclosed tenderness over the position of the scaphoid and marked tenderness upon pressure in the anatomical snuff-box. In attempting motion of the joint there may be such an amount of muscle spasm as to suggest tuberculosis. This was true in one of the cases reported here, when the *x*-ray showed an ununited fracture of the scaphoid with considerable resulting arthritis.

The oldest case in this series was one of fracture of the semilunar, in which pain, tenderness and weakness of the wrist had persisted for thirteen months before operation was resorted to; in this case operation gave a very satisfactory result. Such a case seems to suggest the importance of having an *x*-ray made early in all cases which may at first appear to be sprained wrist.

These cases seem to be difficult to keep under treatment, the patient possibly thinking the injury a slight one is prone to discontinue his visits to the hospital. Of the fifteen cases noted here, from 1914 until March, 1920, six of them disappeared before the end of the first week. Those that continued their visits were kept on an anterior straight splint for periods varying up to eight weeks.

In an exhaustive article by Codman and Chase, in the *Annals of Surgery* for March and June, 1905, their experience indicates that the best treatment for fracture of the scaphoid is as follows: Fixation on a splint for four weeks, then massage for four weeks. After this, if there is no encouraging improvement and the *x*-ray shows no union, they advise excision of the proximal fragment of the scaphoid. Fixation seems not worth attempting after the eighth week if the patient has been using the wrist. Excision of the proximal fragment seems to give better results than the conservative treatment.

Following are the cases found in the dispensary records from 1914 until March, 1920.

Case I.—H. F., automobile mechanic, while in a stooping position and pulling backward on a wrench, slipped and fell backward; throwing back his right hand to break the force of the fall, he struck with considerable force. His wrist became painful, with slight swelling, and he consulted a physician, who diagnosed sprained wrist. He continued at work, the wrist remaining painful on motion; there was no ecchymosis when he presented himself for examination. An *x*-ray showed a fracture of the scaphoid in good position (Fig. 35). The condition was explained to him, but he refused to quit work and have his wrist on a splint. When seen several weeks later, he was wearing a leather wrist band, and stated that although the wrist was weak and in some movements painful, he preferred not to lose any time from work, and he was lost sight of.

Case II.—W. F., aged fourteen years, fell about six feet and landed on his outstretched hand. Came to the hospital complaining of pain in the wrist. The *x*-ray showed an incomplete fracture of the scaphoid bone. He made only two visits and then disappeared.

Case III.—H. S., aged twenty-one years, came for pain in the wrist; no definite history of injury given. *X*-ray showed a fracture of the scaphoid in good position (Fig. 36). Continued his visits until December 23, 1914. An attempt in April, 1920, to locate him disclosed that he had moved away from the city. His relatives said he did not complain of his wrist.

Case IV.—E. F., male, aged twenty-one years, spinner, December 14, 1914, came to the dispensary for pain in the wrist which he had injured in a fight. The disability in this case was such that a provisional diagnosis of tuberculosis was made. An *x*-ray showed a fracture of the scaphoid and was negative for tuberculosis. A straight anterior splint was applied, which the patient wore for two weeks. He removed the splint himself and returned to work and never returned to the dispensary.

Examination April 14, 1920: Wrist showed a prominence on the dorsum over the position of the scaphoid; there is no tenderness; motions of the wrist in all directions are as good as in the uninjured wrist. Says the wrist was painful for a period of six to eight weeks following the injury; now he has no pain except occasionally following a hard day's work.

Case V.—G. Y., male, aged twenty-six years, laborer. While in the supine position and pushing forcibly outward with his hands he experienced a pain in the right wrist. For the last thirteen months it has been treated at various times as a sprain; it always remained weak and at times painful. Examination showed the wrist slightly tender and swollen, with the greatest tenderness one inch above the upper end of the metacarpal of the middle finger. A diagnosis was made of fracture of the scaphoid. The *x*-ray showed a fracture of the semilunar. The wrist was placed on a straight splint for four weeks, followed by massage, with no improvement. Operation was advised.

February 3, 1915. Operation by Dr. Gill. Ether. Skin painted with iodin. Transverse incision on dorsum of wrist. Comminuted fracture of the semilunar found. All fragments removed. Capsule closed with No. I catgut. Skin closed with chromic gut. Painted with iodin and an alcohol dressing applied, and splint. He made an uneventful recovery.

Examined April 11, 1920. Movements of flexion and extension are considerably restricted. There is no pain in the wrist. He is engaged in hard work and says his wrist is entirely satisfactory to him.

Case VI.—T. D., male, aged thirty-two years, teamster, October 28, 1914. Comes to the dispensary complaining of pain in the wrist. A diagnosis of sprain was made. The *x*-ray showed a fracture of the scaphoid. Splint was worn until November 20. Continued visits until January 7, 1915. An attempt to locate him in April, 1920, was not successful.

Case VII.—R. R., aged twenty-one years, October 28, 1914. Came to the dispensary complaining of pain in the wrist. A diagnosis of contusion was made. The *x*-ray showed a fracture of the scaphoid. Continued treatment until December 24, 1914.

Case VIII.—T. O., male, aged twenty-nine years, teamster, January 27, 1914. Pain in the wrist for five days. A diagnosis of sprain was made. The x-ray showed a fracture of the scaphoid bone. Continued treatment until February 7, 1914.

Case IX.—W. Y., male, laborer, March 8, 1916. Marked swelling and disability of the right wrist. Pain in the wrist came on following a fall. The *x*-ray showed a fracture of the semilunar with chipping of the lower end of the radius. Continued treatment until April 29, 1916. An attempt to locate the patient in April, 1920, was not successful.

Case X.—J. G., male, aged twenty-four years, machinist, January 12, 1914. Complains of pain in wrist. *X*-ray showed fracture of scaphoid. He made only two visits.

Case XI.—H. L., aged eighteen years, male. Pain in the wrist for three months. Has been especially tender since a boxing bout recently. Diagnosed sprained wrist. The *x*-ray showed a fracture of the scaphoid. He made only two visits to the dispensary.

Case XII.—J. P., male, aged twenty years, February 14, 1919. He was thrown from a horse in May, 1918, and since then has had pain in the right wrist. *X*-ray shows a fracture of the scaphoid. He made only one visit to the dispensary.

Examined April 11, 1920. All motions of wrist are as good as in the uninjured wrist. Does heavy work and occasionally has pain at night following a hard day. Wears a leather wrist band at times.

Case XIII.—E. J., aged forty-two years, March 4, 1919. Injured left wrist on February 25, 1919. Complains of pain. *X*-ray showed fracture of scaphoid. Continued treatment until April 14, 1919.

Case XIV.—A. S., aged twenty-three years, January 24, 1920. While in the army cranking an automobile in France in May, 1919, he was struck by the handle on the left wrist. He experienced sudden pain in the wrist. This he thought was not sufficient to report at the time. Upon return to civil life he noticed weakness of the wrist, with pain on motion. Examination by *x*-ray showed a fracture of the scaphoid. He made only two visits and then said he could not take time from his work for prolonged treatment or operation, and he returned to his work. When followed up three weeks later the patient was at work and said his wrist was not troubling him enough to take time for proper treatment to be tried.

Case XV.—T. S., aged fourteen years. He fell from a ladder and struck on the right hand. Wrist is swollen and painful, with marked tenderness on the anterior surface in the region of the pisiform bone. Placed on splint. *X*-ray showed a fracture of the pisiform (Fig. 37). He made only two visits.

In only one of these cases (Case IX) was there a fracture of any other bone, and in this there was a slight chipping of the lower end of the radius, with fracture of the semilunar bone. It is to be regretted that many of these cases preferred to put up with a certain amount of disability rather than continue their treatment, and that only one of them consented to operation with excision of the offending fragments. This case gave a very satisfactory result in a wrist that was a constant hindrance for thirteen months preceding operation.

FOUR CASES OF SPONTANEOUS (PATHOLOGIC) FRACTURE

By CLAUDE B. SQUIRES, M.D.
RESIDENT PHYSICIAN IN THE HOSPITAL

The object of this article is to report, briefly, a series of four cases of spontaneous (pathologic) fractures which were treated in the Episcopal Hospital on Dr. Ashhurst's service:

Case I.—Wm. C., aged fifty years.

On December 6, 1919, the patient was struck on the middle finger of the right hand with a piece of iron. Little notice was paid to the injury. There was no break in the skin. An infection developed in the finger and extended to the palm of the right hand, where a palmar abscess developed. Later a cellulitis of the right forearm developed. The middle finger was amputated and later the palmar abscess and cellulitis of the forearm cleared up, but the patient's general condition continued septic, with moderate elevation of temperature, for which no cause could be found. Patient's white blood count was 30,000, red blood count 3,000,000 and hemoglobin 50 per cent. Blood culture was taken a little later and report came back a pure culture of Staphylococcus pyogenes aureus. On January 20, 1920, the patient complained of pain in his right arm. Examination of his arm revealed a *fracture of the right humerus* in about its middle third. This was the first symptom of a fracture and there was no history of injury. There was no swelling or tenderness of the soft parts. *X*-ray examination showed a diseased area extending from 7 to 8 cm. above the middle of the humerus up to its head, with a *subperiosteal fracture* at each end of the diseased area and another fracture high up on the anatomical neck of the humerus (Fig. 38). The diagnosis was at first in doubt, between a metastatic carcinoma, or a quiet osteomyelitis. No primary carcinoma (prostate, thyroid) could be found, however, and as the patient's temperature became normal soon after the fracture, it was concluded that the humerus was the site of metastatic osteomyelitis, probably localized there because of the production of a *locus minoris resistentiæ* by slight recurring trauma in getting the patient in and out of bed to and from his wheel-chair. Soon multiple abscesses developed over the patient's body. February 7, 1920, the patient was operated on for an osteomyelitis of the right humerus and the arm was immobilized by means of a plaster-of-Paris cast. Patient's condition did not improve, and on February 28 the right arm was amputated at the shoulder-joint. Patient's condition still did not improve but seemed to get worse. On March 6 patient was given 500 c.c. of blood by transfusion. Patient died four hours later. Figs. 39 and 40 are from a skiagraph and a photograph made of the humerus dissected from the amputated arm.

X-ray reports by Dr. R. S. Bromer.

January 20, 1920 (2101-D). Upper one-third of the right humerus shows marked rarefaction and absorption. I do not think this is a new growth, but believe it is a chronic osteomyelitis of infectious origin secondary to infection of the hand. The involvement is extensive. There is a line of fracture just at the level of the anatomic neck and another about three inches below it. The fragment between these has the appearance of a sequestrum. It shows no signs of proliferation whatever or increase in calcium deposits often seen in a chronic osteomyelitis. The lack of this suggests a secondary carcinoma, and the possibility of this must be borne in mind.

January 24 (2144-D). Examination of shoulder shows the same condition as previous examination. The fracture at lower end of lesion is more marked at this examination. Examination of right hand shows roughening of the metacarpals and phalanges, but no definite seat of osteomyelitis.

February 18 (2415-D). Examination shows somewhat more displacement of the lower fragment outward. There is no bone regeneration or proliferation.

March 1, (2487-D). Plates of the specimen of the humerus removed from the amputated limb show in clearer detail the condition found on previous examinations. The upper half of the humerus shows rarefaction throughout; the periosteum is raised and shows proliferation. The line of fracture on the middle third is shown. The cortex is very thin and in places has disappeared entirely. There are no signs of sequestration. The picture is that of an acute osteomyelitis.

Case II.—Mrs. E. F., aged sixty-nine years.

Several of the patient's relatives had cancer. Father and two sisters died with tuberculosis.

In January, 1918, the patient noticed a lump in her left breast. She entered the Stetson Hospital and was operated on for scirrhous carcinoma of the breast. She recovered nicely from this and went to work again. In May, 1919, the patient noticed pain in her right foot and knee and a little later pain was noticed in the middle of her right thigh. At this time there was a slight swelling on the anterior surface of the right thigh. Pain in her knee and thigh became worse and patient was unable to work. She entered the Stetson Hospital for treatment. Shortly after the patient was admitted to the hospital her leg was raised by a nurse in order to place a pillow under it. As this was done a sharp snap was heard and patient complained of a severe pain in her thigh. Examination showed a fracture of the right femur at about the middle third. Patient was treated for the fracture and shortly after this was transferred to the service of Dr. Astley P. C. Ashhurst at the Episcopal Hospital. On admission to the Episcopal Hospital a diagnosis of pathologic fracture of the right femur (metastatic carcinoma) was made.

X-ray examinations by Dr. R. S. Bromer.

December 23 (1798-D). Complete absorption of bone at site of pathologic fracture of the right femur.

March 11 (2658-D). There is marked rarefaction at site of fracture and the lower third of the femur, typical of a metastatic condition.

April 17 (3064-D). Examination of the femur (removed post mortem) shows the old fracture of the femur with formation of considerable amount of new bony growth at the site of fracture. This does not have the typical appearance of callus, being much more proliferative, laminated and thickened than callus usually appears. Union seems fair. The portion of shaft above the fracture is much rarefied and has a worm-eaten appearance. It resembles very much the picture of secondary carcinoma.

On December 15, the day after admission the deformity was reduced under nitrous oxide gas and a plaster-of-Paris cast applied to the entire lower extremity and pelvis. After about nine or ten weeks, slight union having occurred, this gypsum case was removed, and the limb placed in a Thomas knee splint.

The patient was in poor general physical condition and this gradually became worse. On April 5, 1920, she died, probably with toxemia and cardiac failure.

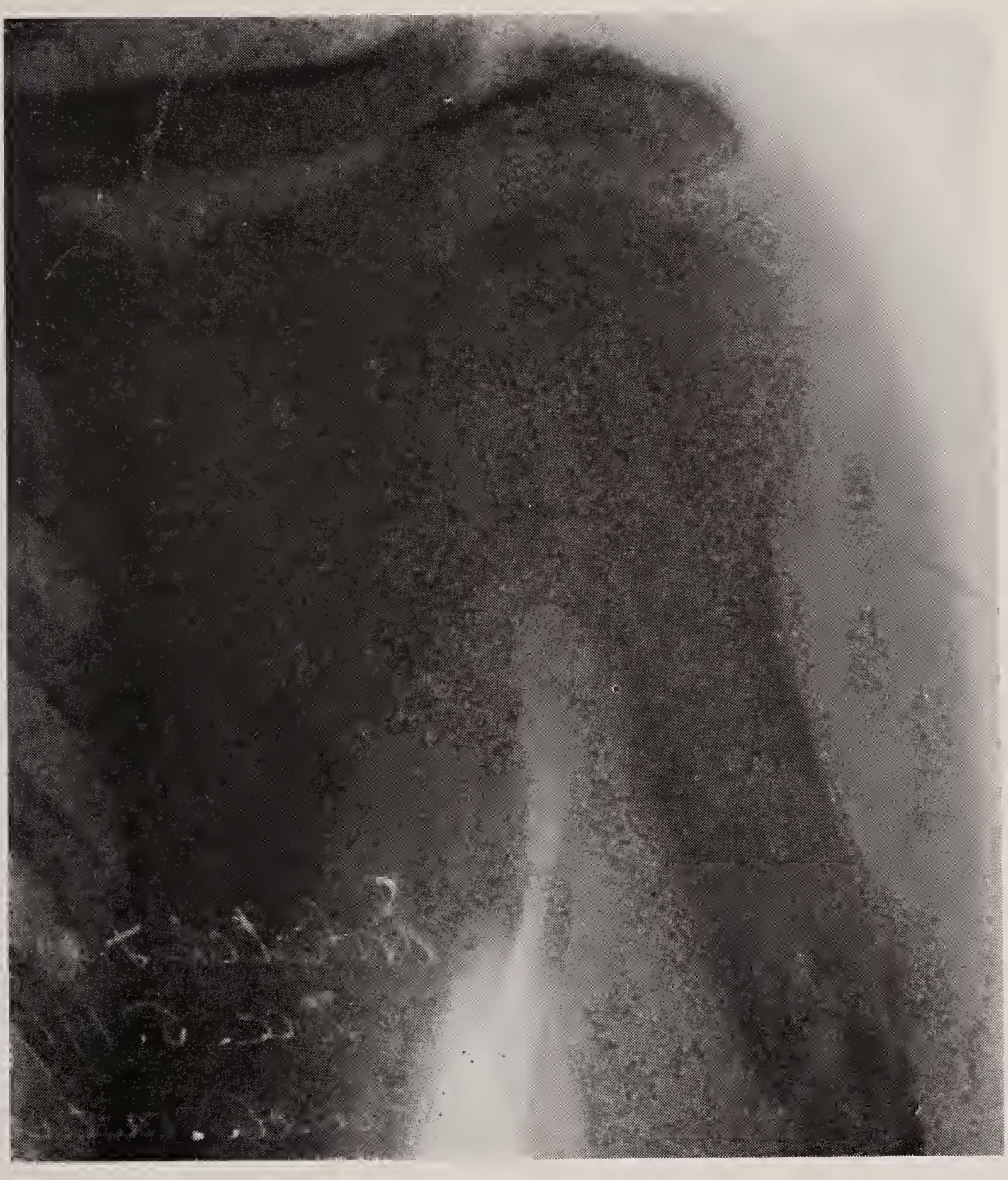

Fig. 38.—Silent osteomyelitis of right humerus, with one pathological fracture above insertion of deltoid, and another at the anatomical neck. (See also Fig. 39.)

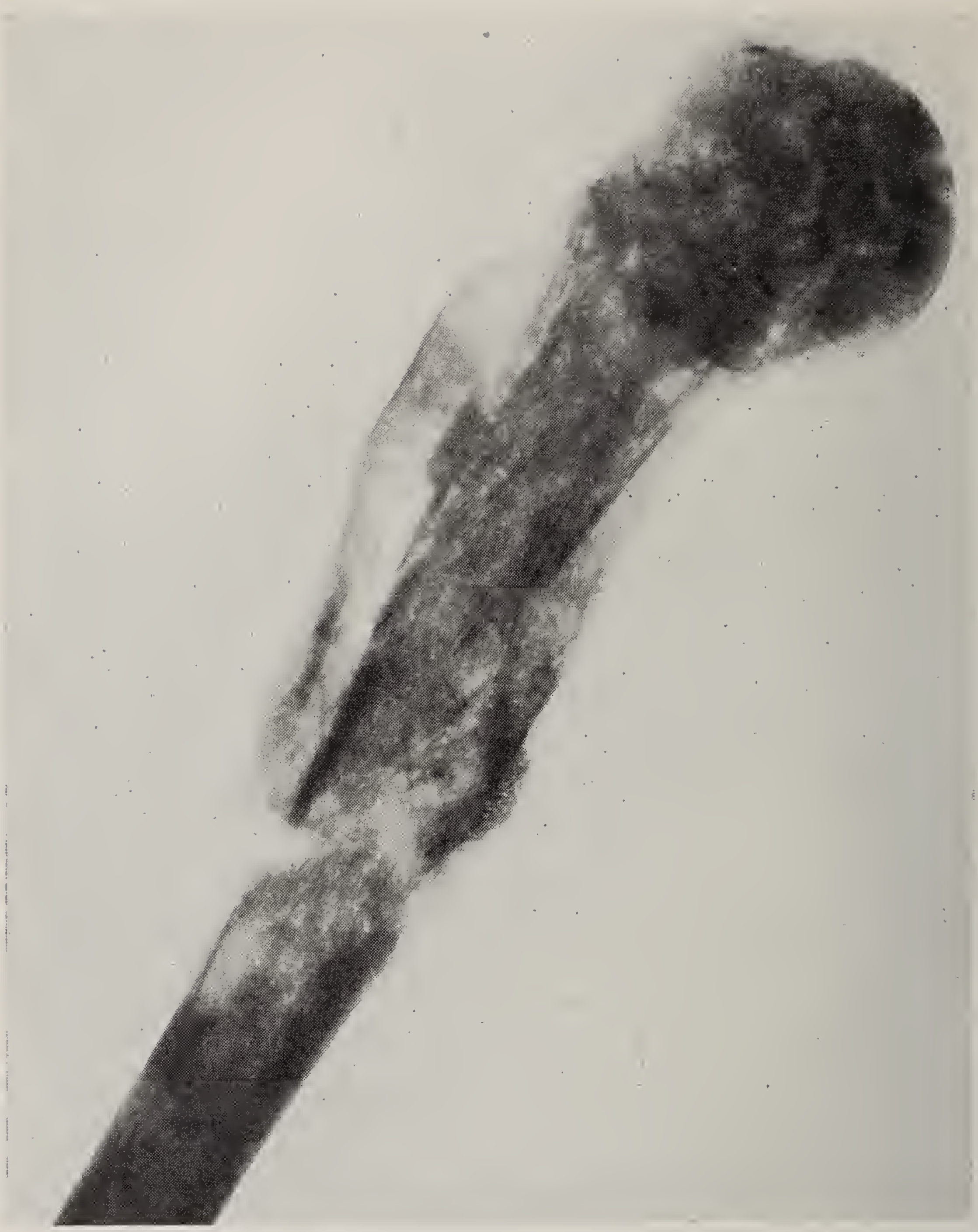

FIG. 39.—Silent osteomyelitis of right humerus, with pathological fractures. Skiagraph of dissected amputation specimen.

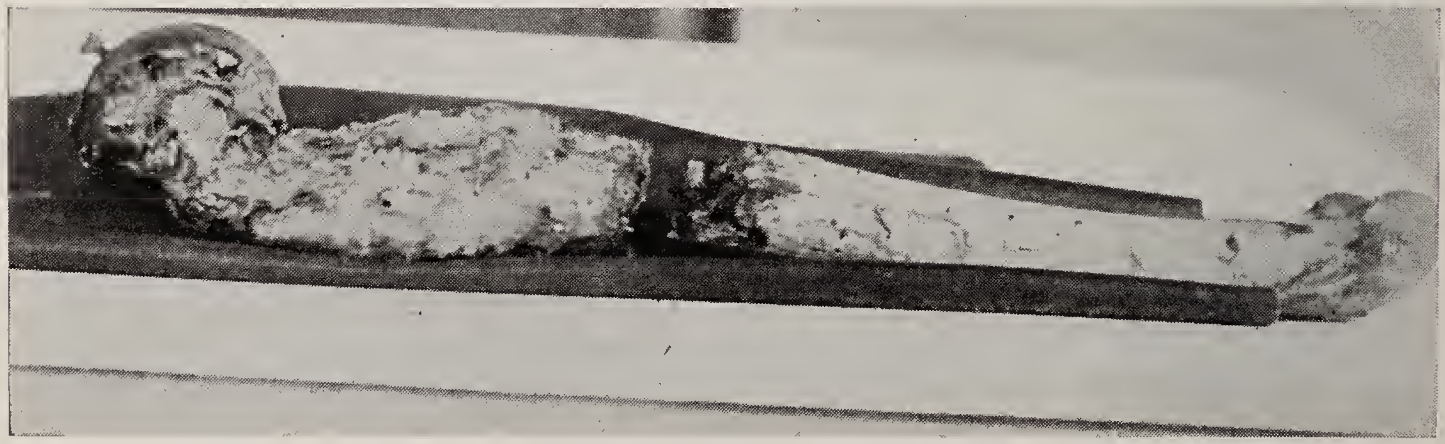

FIG. 40.—Osteomyelitis of humerus with pathological fracture; dissected specimen from amputated arm.

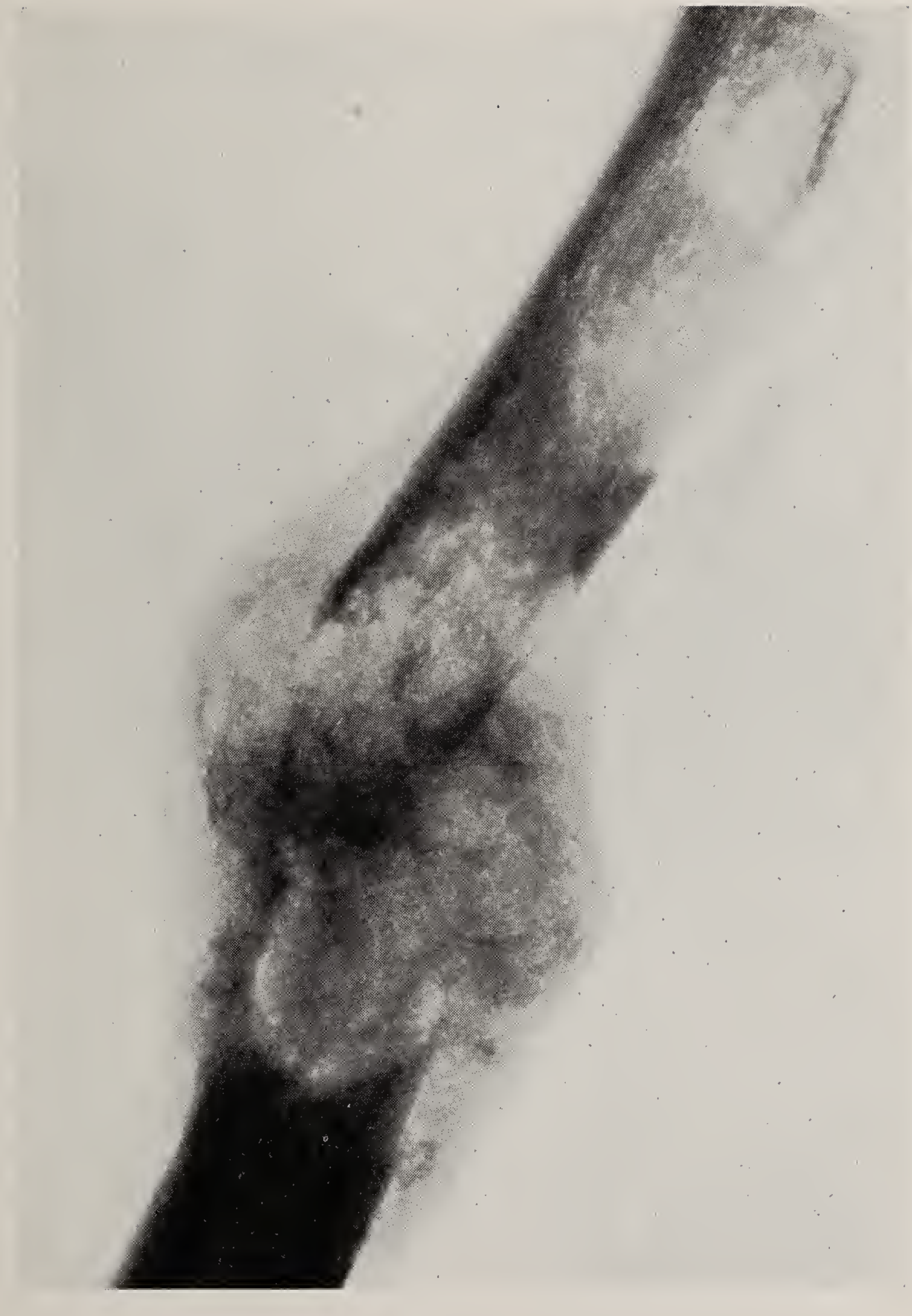

FIG. 41.—Metastatic carcinoma of right femur with pathological fracture (fibrous union). Skiagraph (anteroposterior view) of dissected specimen removed after death. Note atrophy of proximal fragment, infiltrated with carcinoma, and density of distal fragment.

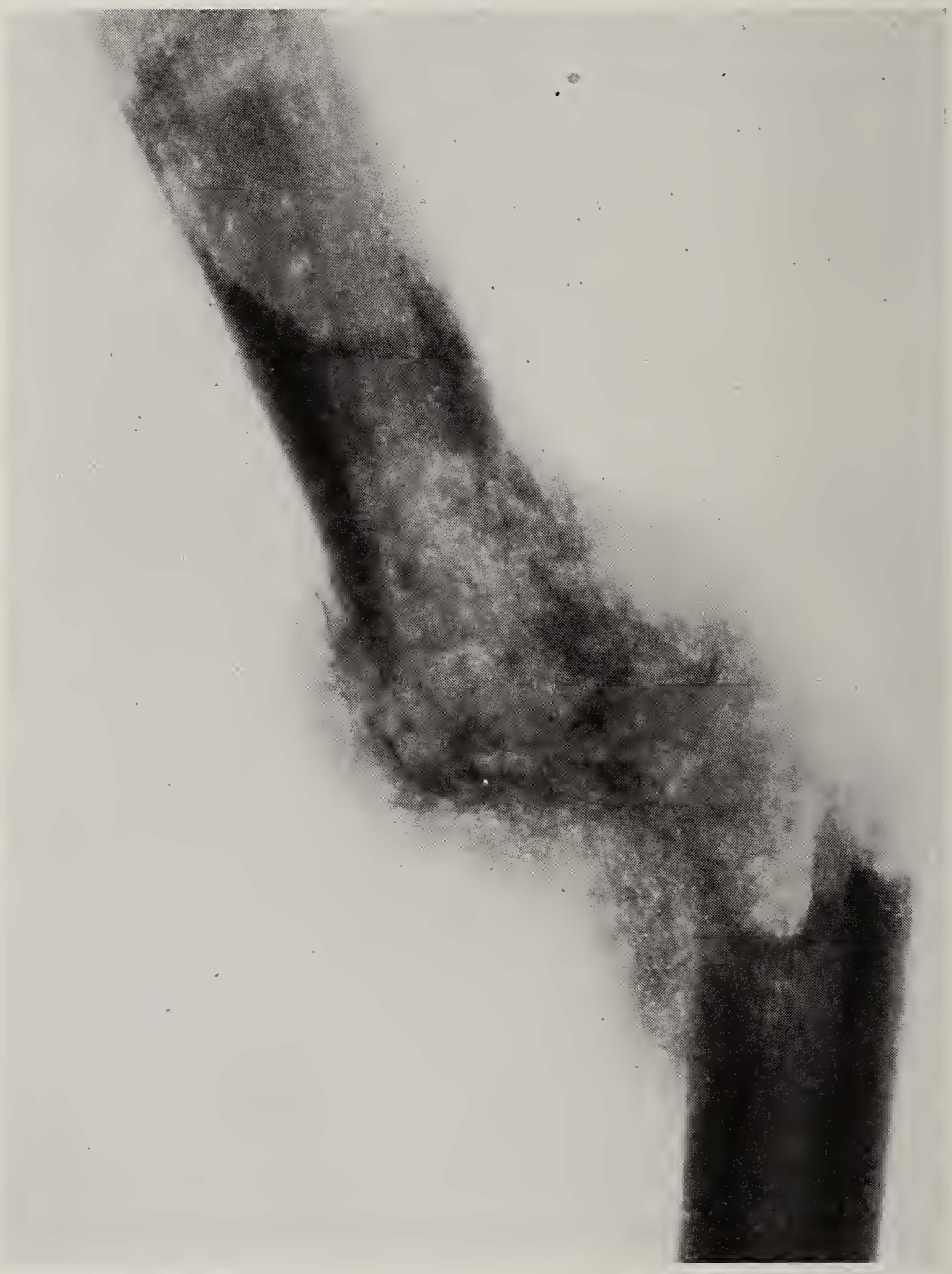

Fig. 42.—Metastatic carcinoma of right femur with pathological fracture (fibrous union). Skiagraph (lateral view) of dissected specimen removed after death. Note the atrophy of the proximal fragment, and the comparatively normal density of the distal fragment.

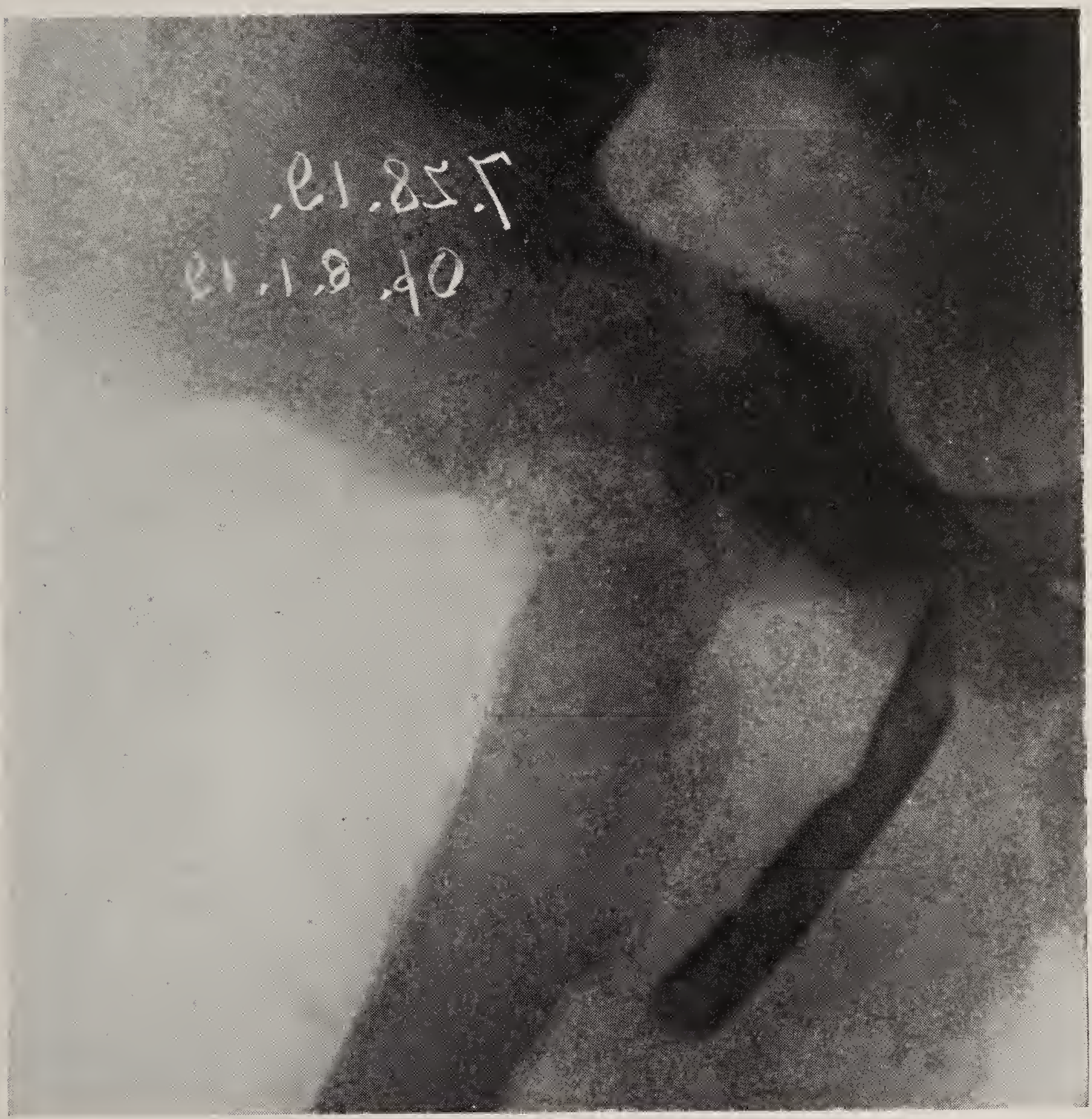

FIG. 43.—Skiagraph of acute osteomyelitis of left femur involving the hip-joint (July 28, 1919, 4 weeks after onset). Drainage tube in adductor region.

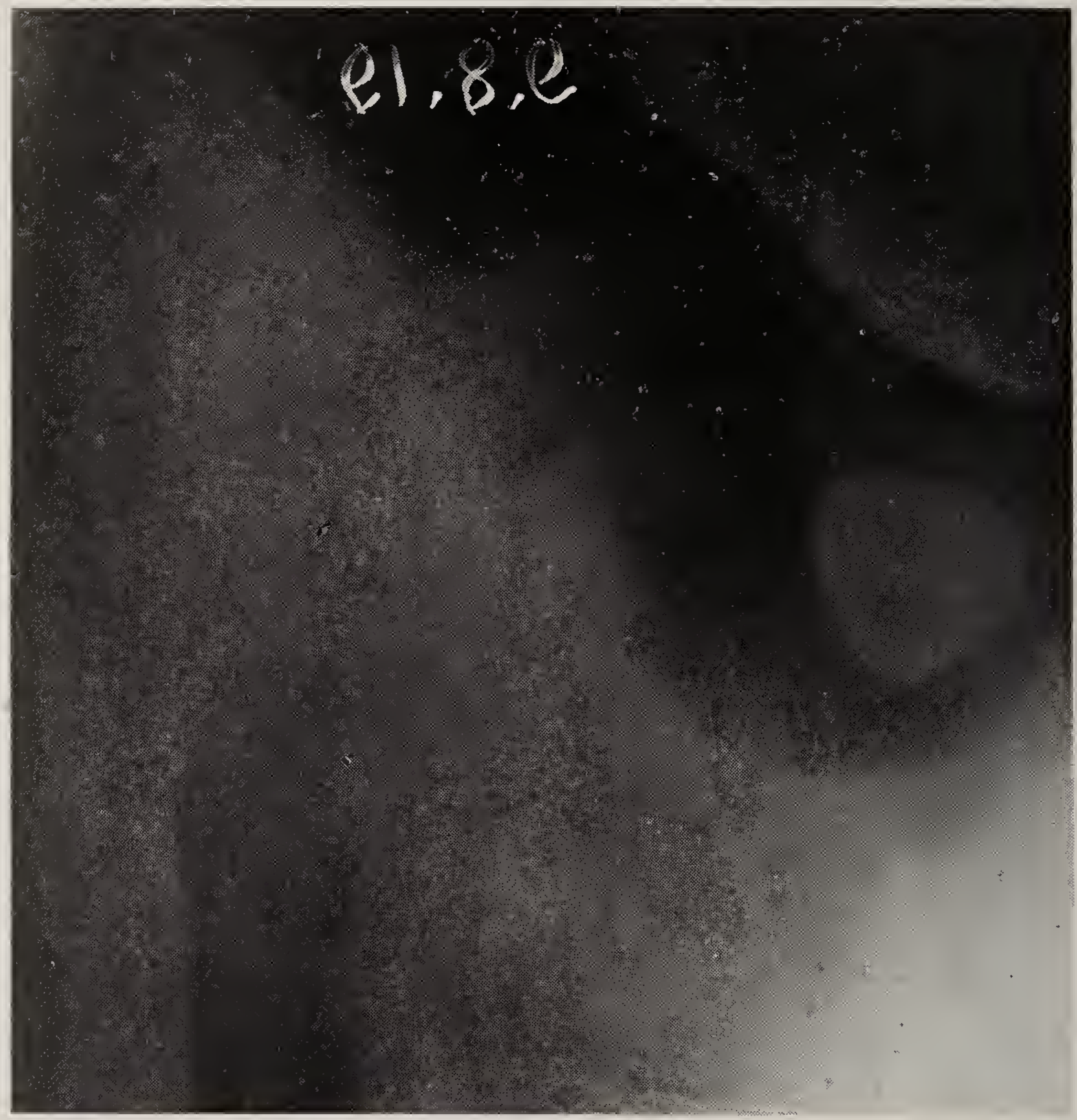

FIG. 44.—Acute osteomyelitis (September 8, 1919), five weeks after excision of hip.

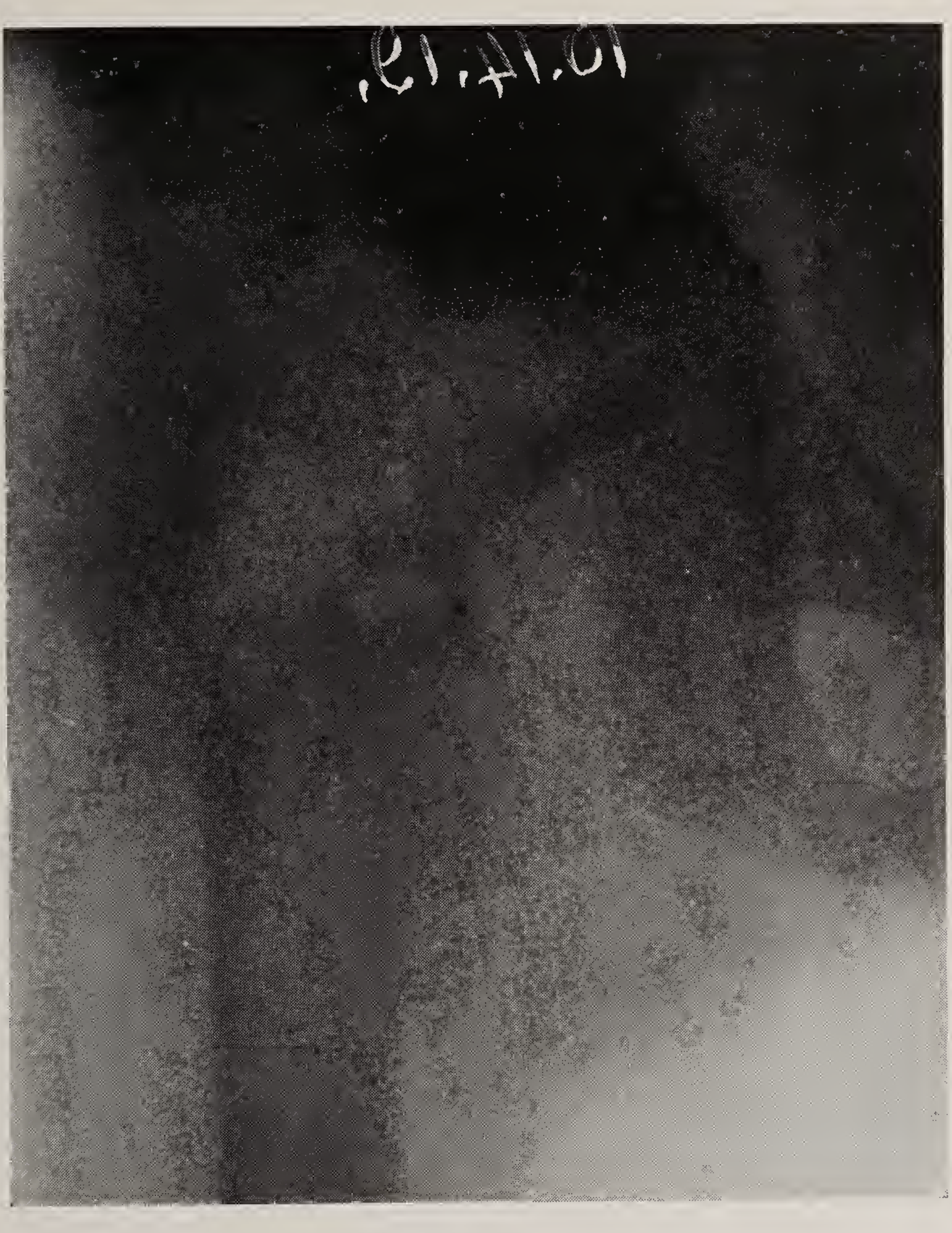

FIG. 45.—Acute osteomyelitis (October 14, 1919) 10½ weeks after excision of hip. Bone reforming.

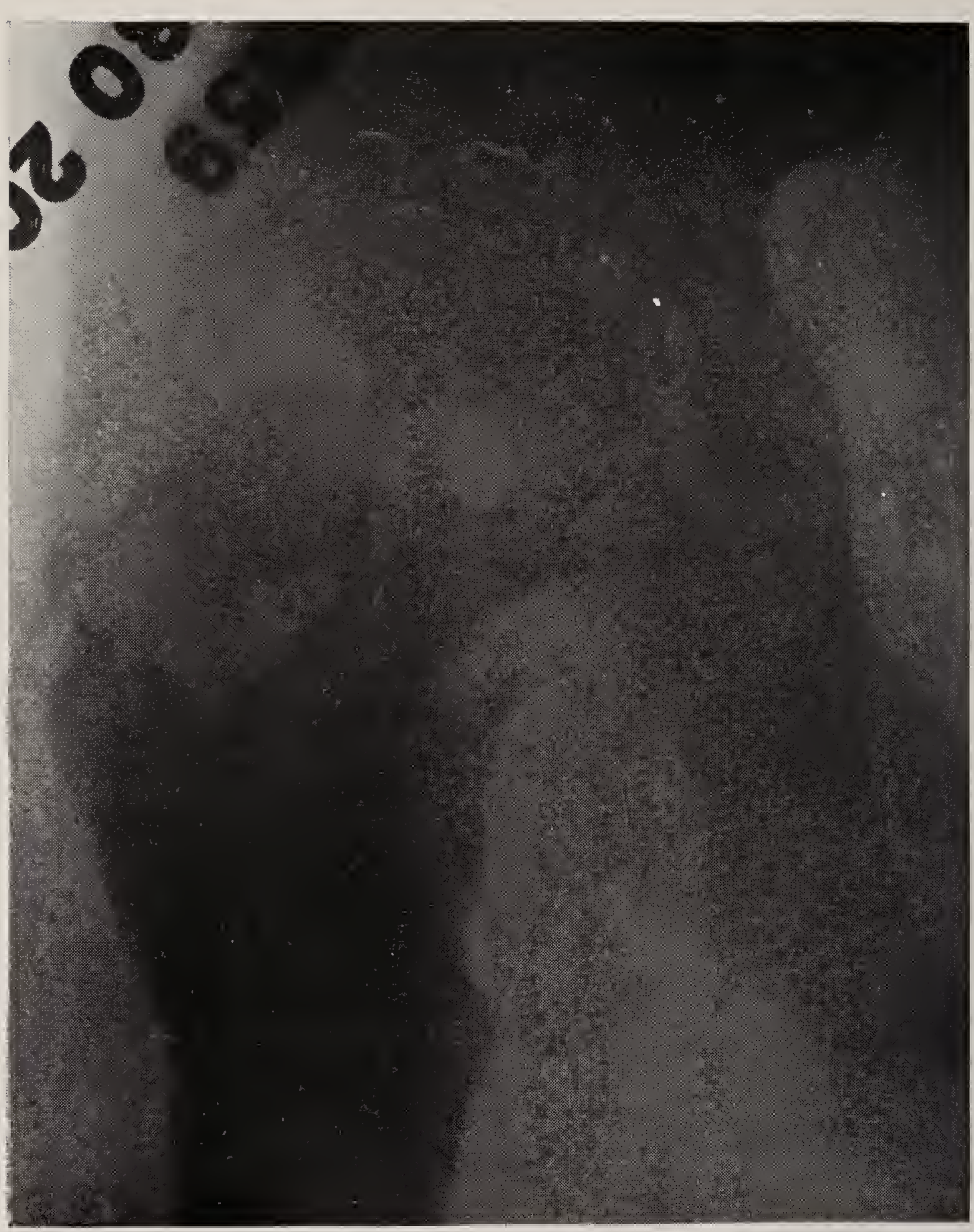

Fig. 46.—Acute osteomyelitis (April 30, 1920) nine months after excision of hip. Stable joint, bone reformed.

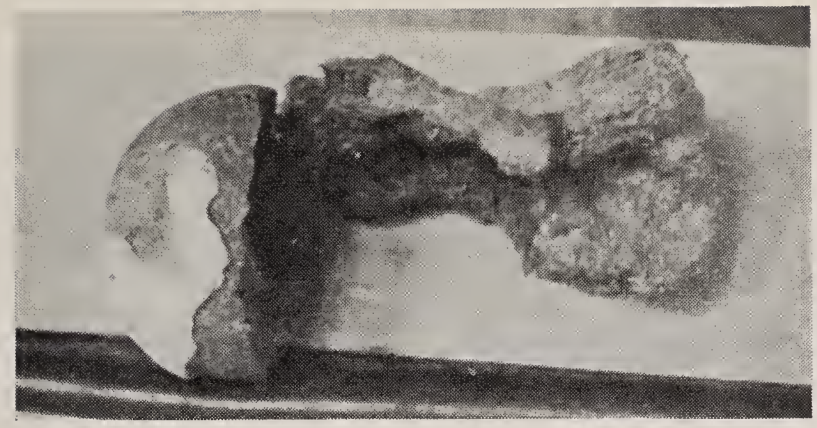

FIG. 47.—Head, neck, and great trochanter of left femur removed by excision of hip for acute osteomyelitis. Pathological fractures between head and neck, and between trochanter and shaft.

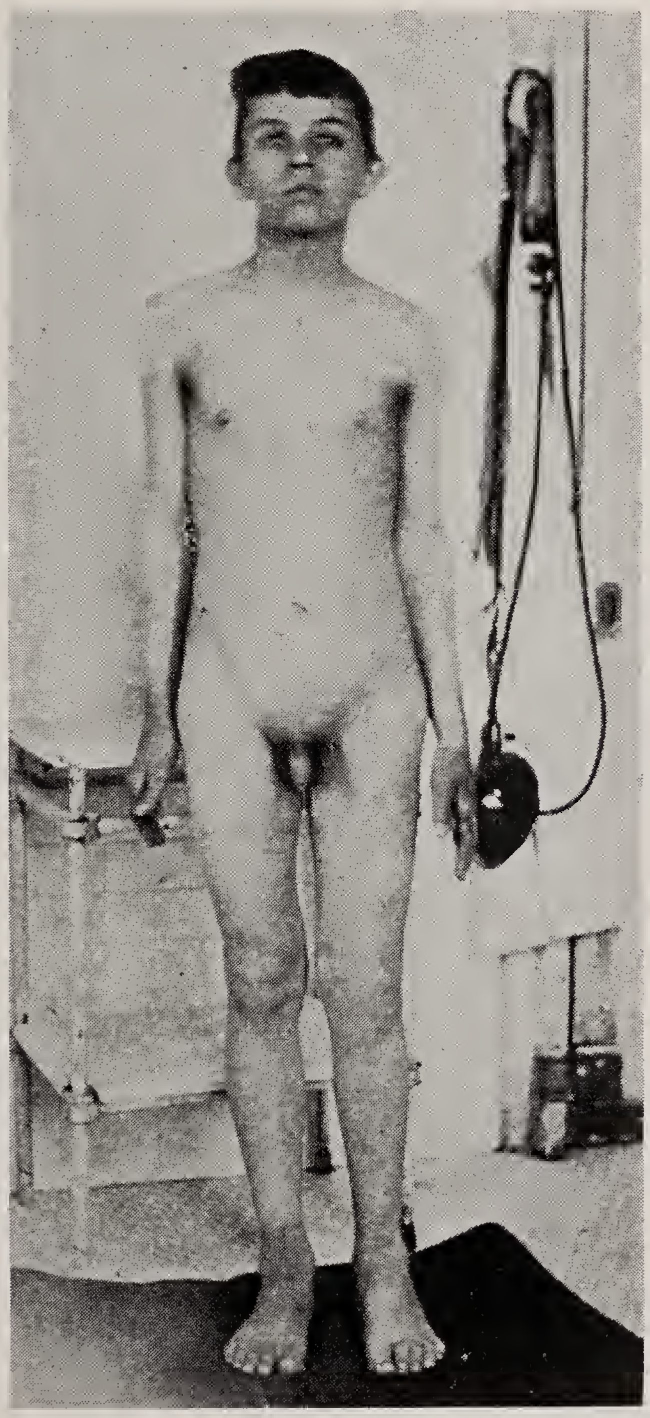

FIG. 48.—Result of excision of left hip for acute osteomyelitis, ten months after operation. Stable joint, 1 cm. shortening, moderate range of movement.

The accompanying illustrations (Figs. 41, 42) are from *x*-rays made of the specimen removed post mortem.

Pathologic diagnosis from Autopsy Report (Dr. C. Y. White).

Recurrent carcinoma of breast.
Carcinoma of axillary glands.
Carcinoma of pleura.
Carcinoma of lung.
Chronic myocarditis.
Chronic interstitial nephritis.
Carcinoma of the periosteum of right femur.
Carcinoma of right femur.
Pathological fracture of right femur.

Case III.—E. V., aged twelve years.

Patient has had a chronic otitis media.

On July 1, 1919, patient noticed pain in his left hip-joint, and on July 12, 1919, there was much swelling and redness about the joint. July 15, 1919, patient was admitted to the Episcopal Hospital (service of Dr. Mutschler), suffering with severe pain in his left hip and an inability to walk on his left leg.

Examination of the left hip and leg on admission to hospital showed a diffusely swollen left hip from Poupart's ligament down to the middle third of the thigh posteriorly, laterally and anteriorly. Severe pain was produced on attempting to move the thigh. Movements of the left hip-joint were limited in all directions. There was marked tenderness all around, but it was most marked anteriorly. The crest of the ilium was very tender. The left knee was flexed but the joint was perfectly normal. There was considerable fever, and the boy appeared very ill.

X-ray examination on July 16, 1919, showed some bone production at the crest of the ilium. Otherwise it was negative.

On July 19, 1919, patient was operated on by Dr. L. H. Mutschler for a cellulitis of the soft structures about the hip-joint, drainage of pus in the adductor region being made.

X-ray examination on July 28, 1919, showed an osteomyelitis of the upper end of the left femur and hip-joint but no fracture (Fig. 43).

On August 1, 1919, patient was operated on by Dr. A. P. C. Ashhurst. An excision of the left hip-joint was done. On opening up the left hip the periosteum was found to be thickened and the great trochanter was found to be detached. Also the head of the femur was found to be loose in the acetabulum, detached from the neck. Pus was found in the hip-joint, in the adductor region and on the exterior surface of femur. The shaft of femur was guttered for 10 cm. below greater trochanter and head and neck of femur removed (Fig. 47). Drainage was put in. Limb placed in Thomas splint, in suspension.

On August 8, 1919, cast was applied to left leg and hip.

Patient recovered nicely and was discharged October 8, 1919, with about 1 cm. shortening of left leg. Was referred to the surgical dispensary of the hospital to have incision, which was still draining a little, dressed daily.

April 14, 1920. Incision on left thigh has healed up and there is about 3 cm. shortening of the left leg. Patient can walk on his left leg with a little limp, which results from the shortening. There is no pain or tenderness about the joint, which is stable, but has very little if any motion. Figs. 44 and 45 show the progress of consolidation after excision of the hip; and Figs. 46 and 48 show the end-result.

Case IV.—Mrs. M. T., aged forty-one years.

Patient entered the Episcopal Hospital's Incurable Ward (Harrison Memorial House) August 3, 1914. On admission complained of shooting pains around her abdomen, headache and difficult defecation. Patient states that she was well until after her child was born in 1902. Six months later was operated on for some ovarian trouble. Child lived eleven months. Husband died with tuberculosis. In 1908 patient began to suffer with sick headache and vomiting. A little while later patient began to have pain in legs and thighs and then in abdomen. The pains were severe and shooting in character, then the girdle pain came on. On admission to the Harrison Memorial House in 1914 patient was thirty-five years of age, had a small bony skeleton, poorly nourished and developed. Arteries and veins negative. Lymph glands negative. Extremities were hypotonic. Eyes: pupils were dilated and did not react to light and very sluggishly to accommodation. Patient has a Charcot joint of left knee. Wassermann test was positive.

In September, 1918, when patient moved to use a bed-pan there was a sudden snap in her left leg. Examination showed a fracture of lower one-third of femur.

Examination at present time, April, 1920, shows a markedly emaciated patient, bedridden and in an extremely poor physical condition. Patient is unable to move and can hardly speak. Examination of patient's left knee shows a large fusiform-shaped joint which has broken down and discharges quite a little pus.

Library

Just above the knee there is a fracture of the femur. It was not possible to get an *x*-ray on account of the poor condition of patient.

April 29, 1920, patient died.

Etiology of Spontaneous (Pathologic) Fractures. In considering the etiology of spontaneous fractures there are two factors which must be considered: (1) Diseased bone and (2) injury. The latter may be in the form of muscular spasm or action, or external violence. A spontaneous fracture may occur without any external violence or with only slight external violence. In Case I there is no history of external violence or muscular spasm, the patient had been lying in bed quietly for about six weeks. In Case II the patient's leg had only been lifted up off her bed when the fracture occurred. In Case III the fracture was not discovered until the patient was being operated on for hip-joint disease; at that time the great trochanter was found to be detached from the femur, and the head from the neck. There was no history of external violence or muscular spasm. In Case IV the fracture of the left femur occurred when the patient raised her leg to use a bed-pan. This did not require much muscular action. Spontaneous fractures have occurred in cases of prolonged muscular spasm. In one case reported both femurs were broken during an epileptic convulsion. In cases of local tetanus the prolonged cramps of muscle may produce atrophy of bone from disuse, leading to spontaneous fracture (Sencert and Carnioley, *Bull. Soc. Chir.*, Paris, 1917–1918). Trophic disturbances also play a large part in spontaneous fractures. Eringhaus reports six cases of tuberculosis of the hip in which there was a fracture of the femur above the condyles. These fractures resulted from atrophic condition of the bones which was due to trophic disturbances.

Local disease of bone plays the most important part in spontaneous fractures. Also general disease such as diabetes, nephritis and tuberculosis must be considered because these conditions may lead to atrophic conditions of bone.

I. *Local conditions* of bone which may lead to spontaneous fractures:

(*a*) Inflammatory changes such as osteomyelitis due to pyogenic organism or tuberculosis or syphilis. In Cases I and III an osteomyelitis pyogenic in origin led up to a fracture. Case IV was due to syphilis.

(*b*) Tumors of bone:

1. Primary sarcoma.

2. Metastatic carcinoma: In Case II the spontaneous fracture of the femur was due to a metastatic carcinoma secondary to a scirrhous carcinoma of the breast. Over one-half of the reported cases of spontaneous fracture due to carcinoma have occurred in the femur.

3. Bone cysts: These may be of several varieties. These may result from broken down sarcoma or may result from an echinococcus infection or they may be a simple bone cyst.

4. Metastatic hypernephroma or metastatic adenosarcoma from the thyroid.

(*c*) Osteomalacia or mollities ossium frequently leads to multiple spontaneous fractures.

II. There are many *general conditions* such as diabetes, nephritis, tuberculosis, rickets, scurvy, etc., which lead to atrophy and fragility of bone, and consequently a fracture. Also there are many nervous conditions which lead to atrophy and fragility of bone. In going through any large nervous or insane hospital, such as the insane wards of the Philadelphia General Hospital, many cases of spontaneous fracture are found. These fractures occurring in nervous and insane people do not necessarily mean that the patient has been violent or has been abused. A majority of them have occurred on account of fragility and atrophy of bone. The fragility and atrophy of bone is most likely due to trophic changes or disturbances which are brought about by changes in the spinal cord. These changes are seen quite frequently in cases of tabes dorsalis and syringomyelia and spontaneous fractures are of frequent occurrence in these conditions. The fracture may be the first symptom of either condition but as a rule the fracture occurs late in the course of the disease. In Case IV, reported in this article, the patient had tabes dorsalis and had had the condition for about eight years. The fracture of her femur occurred in September, 1918. This patient also had a Charcot joint of her left knee.

In paralysis agitans bones are thought to break easily. A large number of paretic dementias have shown unnatural brittleness and softness of bone. Also the bones of maniacs are frequently fragile and break easily.

Slight tripping, jolts, jars and missteps often cause fracture of the femur in old people and in patients convalescing from a long illness.

Atrophy of bone quite often occurs as result of pressure due to aneurysm. An aneurysm may compress the ribs, sternum or vertebræ and cause atrophy which may lead to a fracture.

SIGNS AND SYMPTOMS OF SPONTANEOUS FRACTURES. A spontaneous fracture may occur with only a very few signs or symptoms. The symptoms may be so slight that a fracture may go unnoticed for a long period of time. When the fracture occurs the patient may say that a snap was heard or that there was sudden severe pain or there may not have been any pain at all. There may be a history of some slight injury or muscular spasm or action.

1. *Pain.* There may be pain when the fracture occurs or there may be no pain at all. In Case I the pain was only slight and the fracture was probably unnoticed for some time. In Case II the pain was marked. In Case IV no pain was produced when the fracture occurred. Pain may be the only symptom, as in Case I. This led to an examination of the patient's arm. The pain is usually made worse by pressure or movement of the part. In syphilis and rickets pain is absent or very slight.

2. *Loss of Function.* This usually exists, especially in fractures of the leg.

3. *Swelling* usually occurs. Often the swelling is absolutely painless.

4. *Deformity.* This may be in any direction. Quite frequently it is entirely absent. It may be due to swelling or to displacement of the fragments. In cases I and III there was no deformity. In Cases II and IV there was marked deformity.

5. *Abnormal mobility* usually is present but mobility may be normal.

6. *Crepitus* is usually found.

7. *Ecchymosis* does not occur in all cases.

DIAGNOSIS. This is based on the history and signs and symptoms and upon *x*-ray examination. When a patient suffering with diseased bone or any chronic disease, or when a bed-ridden or aged patient complains of pain and tenderness of a bone, the part should be examined closely and an *x*-ray picture should be made. History of any slight injury should be gone into carefully.

TREATMENT OF SPONTANEOUS FRACTURES. Briefly the treatment consists in reduction of the deformity under narcosis and immobilization of the part, preferably by the application of a plaster-of-Paris cast or splint. This applies especially to those cases which occur in the aged. In cases of spontaneous fractures secondary to an osteomyelitis due to pyogenic organisms and tuberculosis, the osteomyelitis should be treated just as if there were no fracture, but at the same time the fragments of the fracture should be placed in good apposition and the part immobilized. In Case I the fracture was secondary to an osteomyelitis of the humerus and the latter was guttered and drained and the part immobilized by means of a plaster-of-Paris splint. Later an aëroplane suspension splint was substituted for the plaster-of-Paris splint in order that the arm could be dressed more easily.

In cases due to syphilis the patient should be put on an antisyphilitic treatment and the part immobilized as in any fracture. Osseous union usually takes place in this type of fracture.

In fractures occurring secondary to a tumor of bone the general physical condition, age of patient and the type of tumor have to be considered.

In cases of bone cyst a curettage and immobilization of the part may be all that is necessary, or if the condition recurs a more radical measure, such as resection of a part, could be done. This could be followed by some operations for filling in the defect.

In spontaneous fractures secondary to sarcoma and carcinoma a resection of the part or a radical amputation should be done, provided the general physical condition of the patient warrants such. In case the patient is unable to stand a radical operation the fragments should be brought in as good alignment as possible and a plaster-of-Paris cast applied. Case II could not stand an operation and her right leg was immobilized by means of a plaster-of-Paris cast. This gave her comfort for about eight weeks, and then it was taken off and a Thomas splint was applied. At this time there was apparently fibrous union of the parts.

Patients with spontaneous fractures suffering with general diseases, such as tuberculosis, syphilis, nephritis, diabetes, scurvy, rickets, etc., should be treated just as such a case is generally treated. The general health of such patients should be guarded closely, and they should have plenty of nutritious food, fresh air and sunlight, and should be cared for in a most careful and sanitary way.

A CLINICAL LECTURE ON THE DIAGNOSIS AND TREATMENT OF TUBERCULOUS BONE AND JOINT DISEASE IN CHILDREN[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

1. Pathology. You have heard enough, in the previous lectures of this course, to know somewhat of the pathogenesis and morbid anatomy of tuberculosis in general, and I can be very brief in what I have to say on these topics. The tubercle bacilli lodging, as they usually do, on their first entrance into the body, either in the cervical, bronchial or mesenteric lymphnodes, lead there a peaceful and usually overlooked existence. They may and probably do, from time to time, if these nodes caseate and rupture, escape into the general circulation, but usually perish because in their journey they find no suitable soil for their continued existence. If all goes well they may not give evidence of their existence until adult life. But if somewhere in the body there is established in suitable soil a *locus minoris resistentiæ*, the bacilli in their journeys through the circulation eventually reach this area, find surroundings suitable for their existence, establish themselves in a new home and thrive—they occupy the land, and like the patriarchs and their progency of old, become as the sea sands in multitude.

It has been pointed out by Ely (1911) that the most favorable soil in bone for the development of tubercle bacilli is one in which lymphoid cells exist, such as form the synovial lining of joints; or red bone-marrow, such as exists in children in the cancellous ends of bone, and in short bones of the carpus and tarsus. It thus happens that emboli of tubercle bacilli may occur in all parts of the body, but except in such favorable localities as those just mentioned the bacilli will not survive; and even in favorable soil they may not survive unless the natural resistance of the part is already damaged. It is thus that you find, in about one-third of cases, a history of traumatism, usually comparatively slight, preceding, by a period of several weeks, the development of symptoms of tuberculous disease. Now it is very well known that all children sustain slight joint injuries from time to time, yet comparatively few develop tuberculous arthritis; so that clinical experience, *â posteriori*, confirms what we should expect, *â priori*, from our knowledge of morbid anatomy; and it is necessary to assume, even if it cannot be demonstrated clinically, that the child with tuberculous bone or joint disease has a primary tuberculous focus elsewhere in the body.

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[1] Delivered in the Post-Graduate Course of the Philadelphia Pediatric Society, at the Children's Hospital of Philadelphia, December 12, 1919. Reprinted from *International Clinics*, 1920, XXXth series, vol. ii, p. 1.

The injury which precedes the tuberculous joint symptoms rarely is severe; fractures scarcely ever are followed by tuberculosis and fractures in the tuberculous heal normally. Two explanations are offered for this: one is that the more severe injury arouses better defensive action on the part of the patient; the other is that severe lesions require careful and prolonged treatment, and healing therefore is more liable to occur than after a trivial injury which often is neglected.

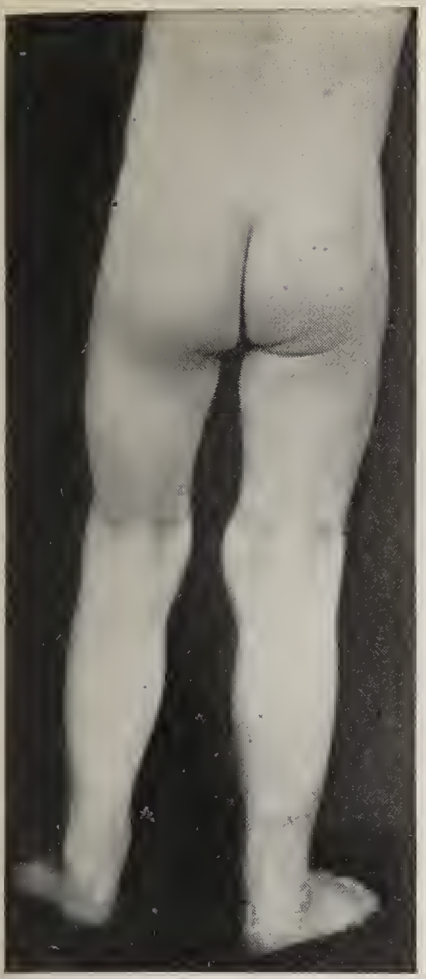
So you will find that the parents bring the child to you complaining of pain and disability; but unless you are more observant than the parents you will often overlook the true site of the disease because the pain is usually of the type known as *referred pain.* There is scarcely a year passes that I do not see one or more little children who have been treated symptomatically by their family physician for weeks and sometimes for months for pains in their stomach, when a cursory examination of their back shows them to be suffering from tuberculosis of the spine, and demonstrates that the belly ache was merely pain referred along the intercostal or lumbar nerves. So, too, with disease of the hip-joint the pain may be referred to the knee, along the obturator nerve, and all the embrocations and liniments which for weeks have been applied to the knee have been unavailing. Stiffness of the joint may have been noted by the parents for some days or weeks, apparent when the child first gets up in the mornings; such stiffness often wears away during the day, but toward evening the joint again becomes disabled, and this is evidenced by slight limp and complaints of pain. Pain may be almost absent except when a joint is used; but frequently a joint which is painless when the child is awake will trouble it at night, causing restlessness; and on falling asleep and relaxing its muscles the child will experience "starting pains," which will rouse it momentarily from sleep with a "night cry." Along with these symptoms you will usually find a slight constant elevation of temperature.

2. Diagnosis. Children suspected of tuberculous joint disease should be examined stripped. Nothing hinders so much proper appreciation of deformity or limitation of joint motion as the presence of clothing. And in examining children, as you know, it is first of all necessary to win their confidence; nothing so quickly repels them, especially if in pain, as abrupt, rough or brutal manners.

Inspection, even in early cases, frequently is enough to indicate the diagnosis. The child will be able to walk to and fro, and you can observe limp, evidences of deformity or other disability. In many cases of tuberculosis of the spine the child, though able to walk, is unwilling to do so because of pain, and will support himself on each piece of furniture he encounters, and when standing at rest may hold his thighs with his hands to aid in supporting his back. Have the child stoop to pick up something off the floor: if there is spinal disease you will often see him bend his knees and his hips, bringing his buttocks almost into contact with the floor until his hand can reach the object, rather than flex his spine to stoop forward normally. In the standing position in early cases of hip disease you will notice, attentively inspecting the child from behind, that there is flattening of the affected buttock, with disappearance of the normal gluteal fold on the same side; and later, when you begin your physical examination, you will find that the diseased hip is held persistently in a slightly flexed and abducted position, a posture which accounts for the changes in outline just noted.

The *physical examination* of the suspected joint gives characteristic evidences of disease. There is spasm of the muscles around the joint, or at least limitation of motion in one or more directions. It becomes incumbent on you, therefore, to know the limits of normal movement, and to exclude in your examination compensatory movements which may be made in adjacent joints—such especially as movements in the pelvic and lumbar joints which may simulate motions in the hip-joint and motions between the scapula and trunk which simulate movements in the shoulder joint. The surest way to ascertain limitation of motion is to compare the movements of the affected joint with those of the corresponding healthy joint. In the case of suspected *hip disease*, have the child recumbent, and, beginning by testing the movements of the normal hip, show him by your manner that you do not intend to cause him any pain; you will then find that very rarely is any resistance aroused. Test first the rotation of the hip by placing the hand flat on the thigh above the knee, and rolling the limb in and out: try the same maneuver on the diseased side; almost without exception the excursion will be less on the affected side. Then try abduction of the normal hip by carrying the limb outward in the coronal plane; attentively watch the anterior-superior spines of the iliac bones and you will find that although they do not move when the normal hip is abducted, yet when an attempt to abduct the affected hip is made it will be evident that the apparent motion of abduction is not occurring in the hip but in the lumbar spine, the pelvis tilting down on the healthy side and up on the side of the diseased hip when attempts to abduct the latter are made. Note next that the child's lumbar spine is not lying flat on the table but is slightly arched; now as you flex the healthy hip until the thigh comes almost into contact with the abdomen you will observe not only that this lumbar lordosis becomes obliterated as you, by hyperflexion of the hip, flex the pelvis on the spine, but also that the thigh on the diseased side rises from the table and the diseased hip is seen to be held in a position of flexion by muscular spasm. On the contrary, when you gently flex the diseased hip as far as can be done without pain the lumbar lordosis similarly disappears but the healthy thigh does not rise from the table, because as the pelvis becomes flexed on the spine there is the possibility of hyperextension in the normal hip, whereas in the diseased hip even full extension is impossible. Next, with the hip flexed to a right angle, test the abduction of the hip in this position: on the normal side you will find the thigh can be brought outward until the outer side of the knee nearly or quite touches the table, without any movement occurring in the pelvis; but on the diseased side abduction in this position is greatly limited. Finally, turn the child over on his stomach, and also in this position test the range of rotation in each hip by bending the knee, and turning the limb in and out; and test for hyperextension, by gently raising the flexed knee from the table; on the normal side the knee can be raised a hand's breadth or thereabouts from the table, whereas on the diseased side, where even normal extension is absent, it may be impossible to raise the knee at all. A confirmatory sign in tuberculosis of the hip, which should not be overlooked, is enlargement of the lymph nodes along the external iliac artery.

In a case of suspected *vertebral tuberculosis*, first examine the child standing: have him bend forward as far as possible, and note if on straightening up again he does so irregularly, by jerky movements, or easily and quickly. Of course, a typical "hump" reveals the true nature of the disease almost



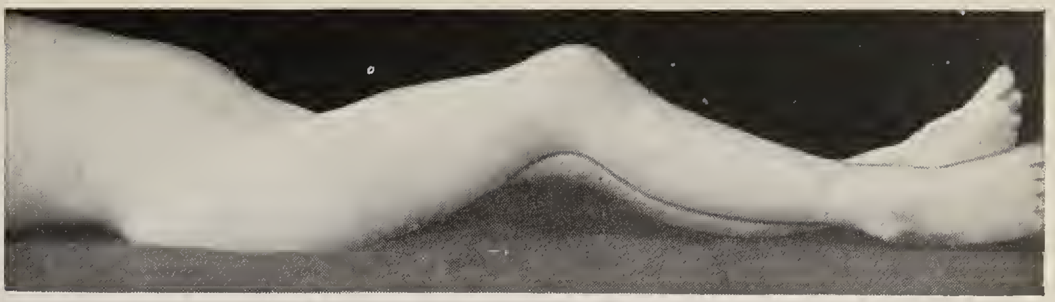


Fig. 50.—Tuberculosis of right hip—persistent flexion of hip causing lordosis of spine when attempt is made to extend the hip. Age 3 years. Duration 9 months.

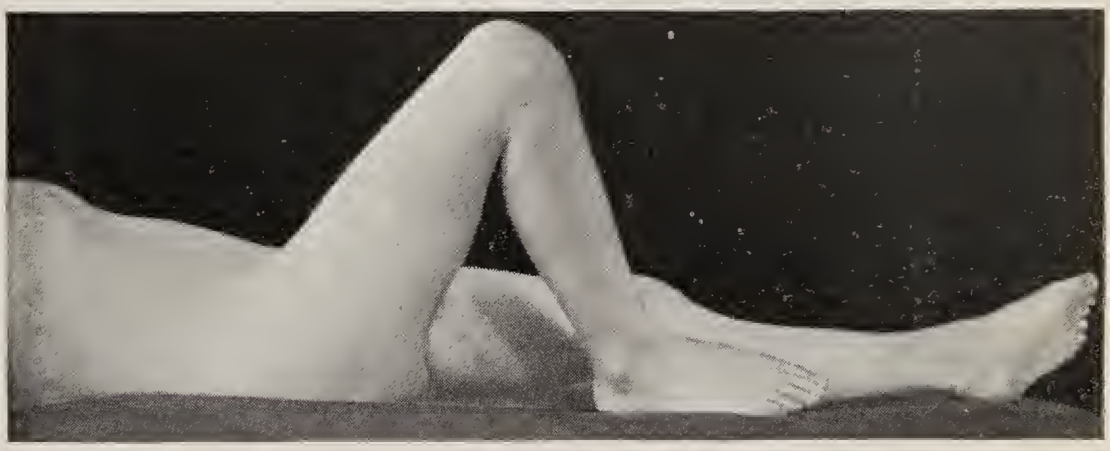
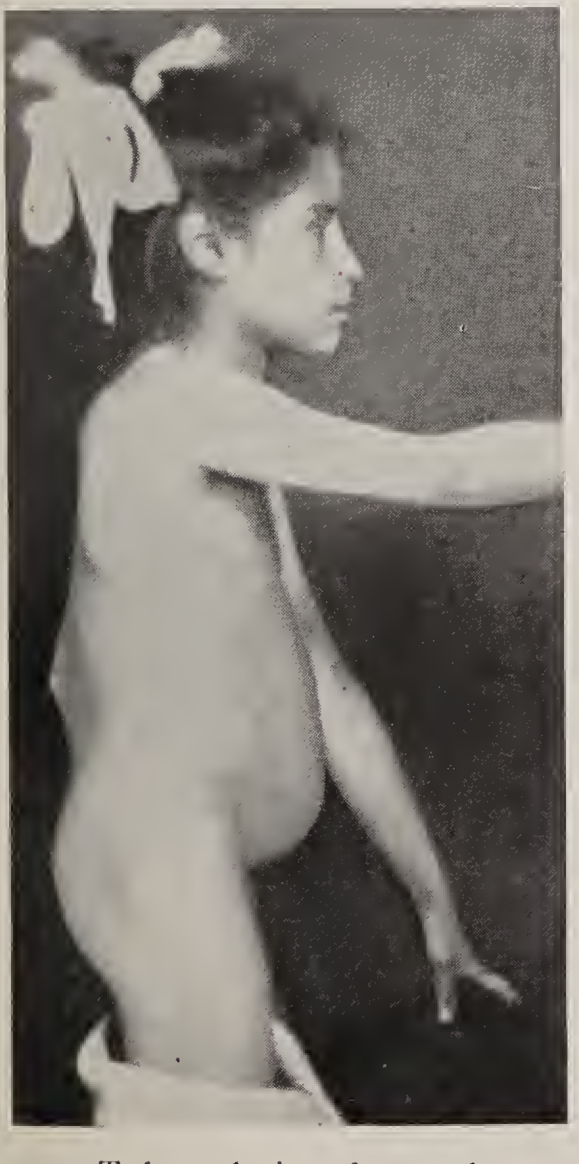


Fig. 49.—Early stage of tuberculosis of left hip. Abduction and flexion causing flattening of buttock and obliteration of the gluteal fold. Age 5 years. Duration 2 months.

Fig. 51.—Tuberculosis of right hip—same patient as in Fig. 50; when the thigh is raised the lumbar lordosis disappears.



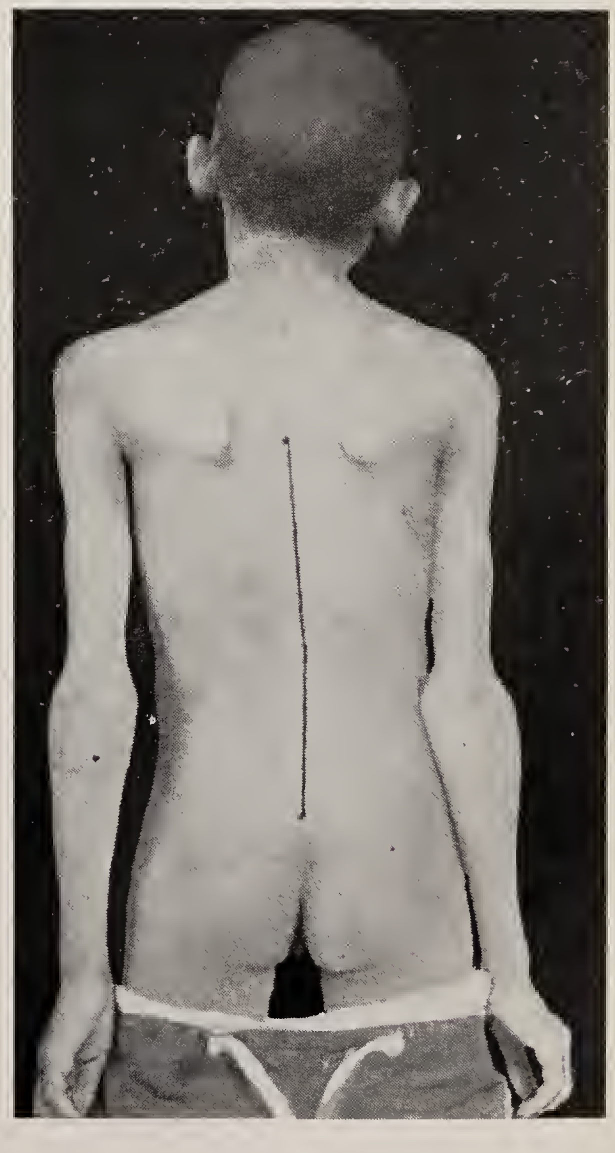


Fig. 52.—Tuberculosis of vertebræ. Age 7 years. Duration 1 year. Showing angular kyphos.

Fig. 53.—Tuberculosis of vertebræ, showing lateral deviation to left. Age 13 years. Duration 7 months.

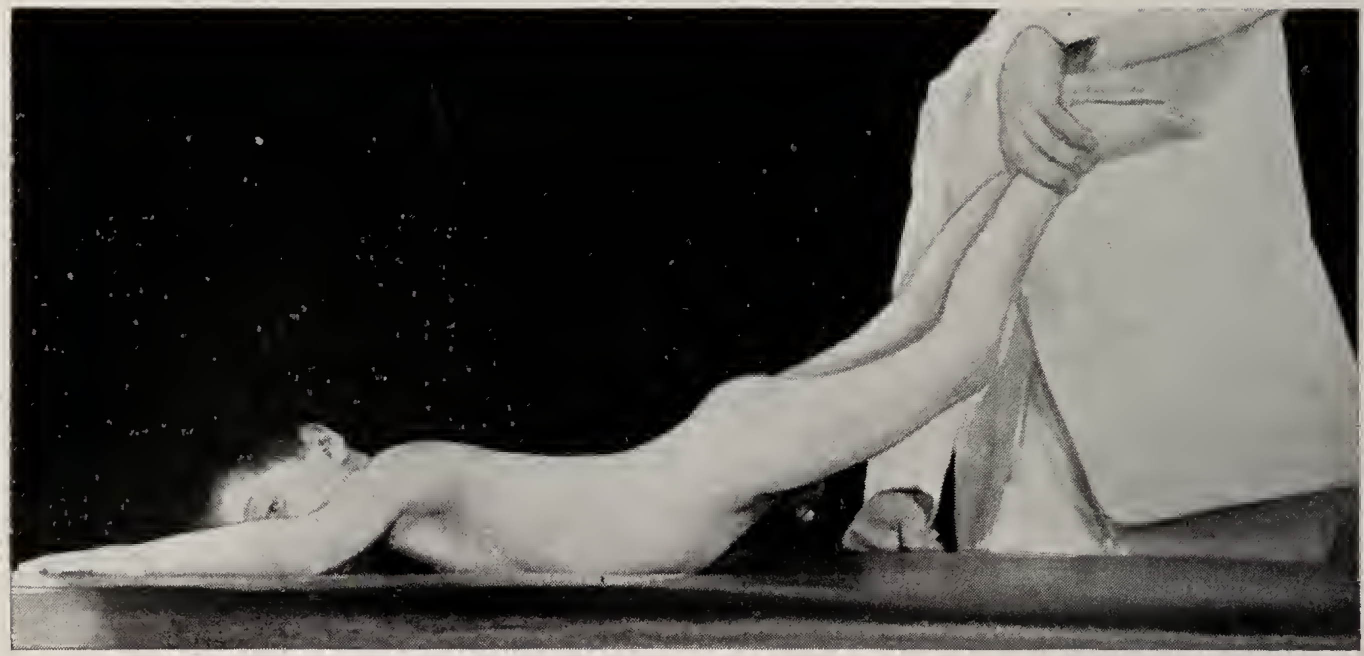


FIG. 54.—Tuberculosis of vertebræ; testing the hyperextension of the spine.

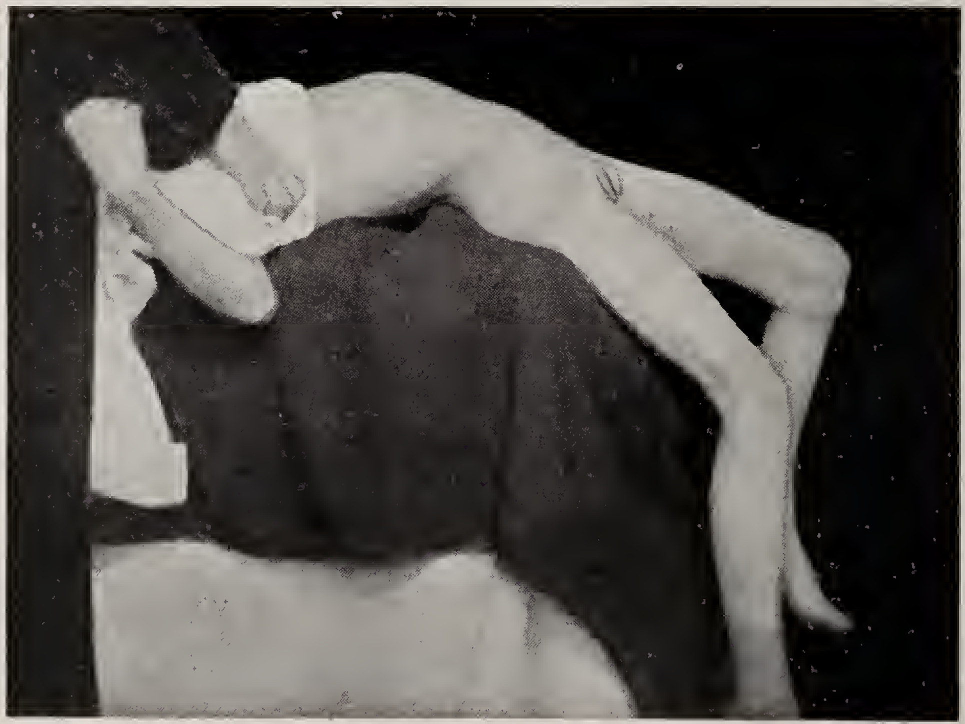


FIG. 55.—Tuberculosis of the vertebræ; contracture of left psoas muscle from abscess. The right hip shows normal hyperextension. Age 11 years. Pott's disease for 4 years; abscess for 8 months.

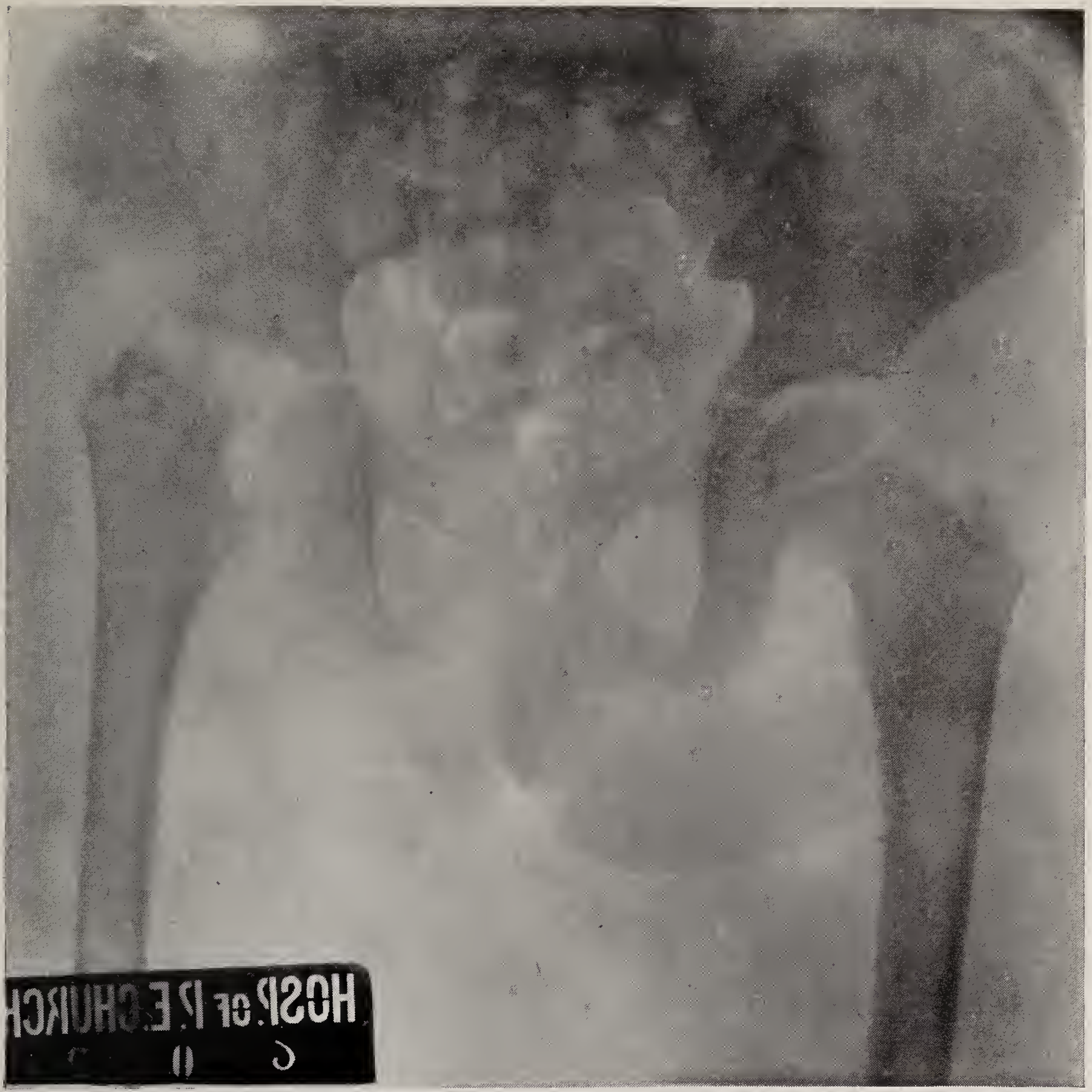


Fig. 56.—Tuberculosis of the hip with abscess; head and neck of femur destroyed.

Fig. 57.—Bradford's gas-pipe frame for treatment of tuberculosis of the joints in recumbency.

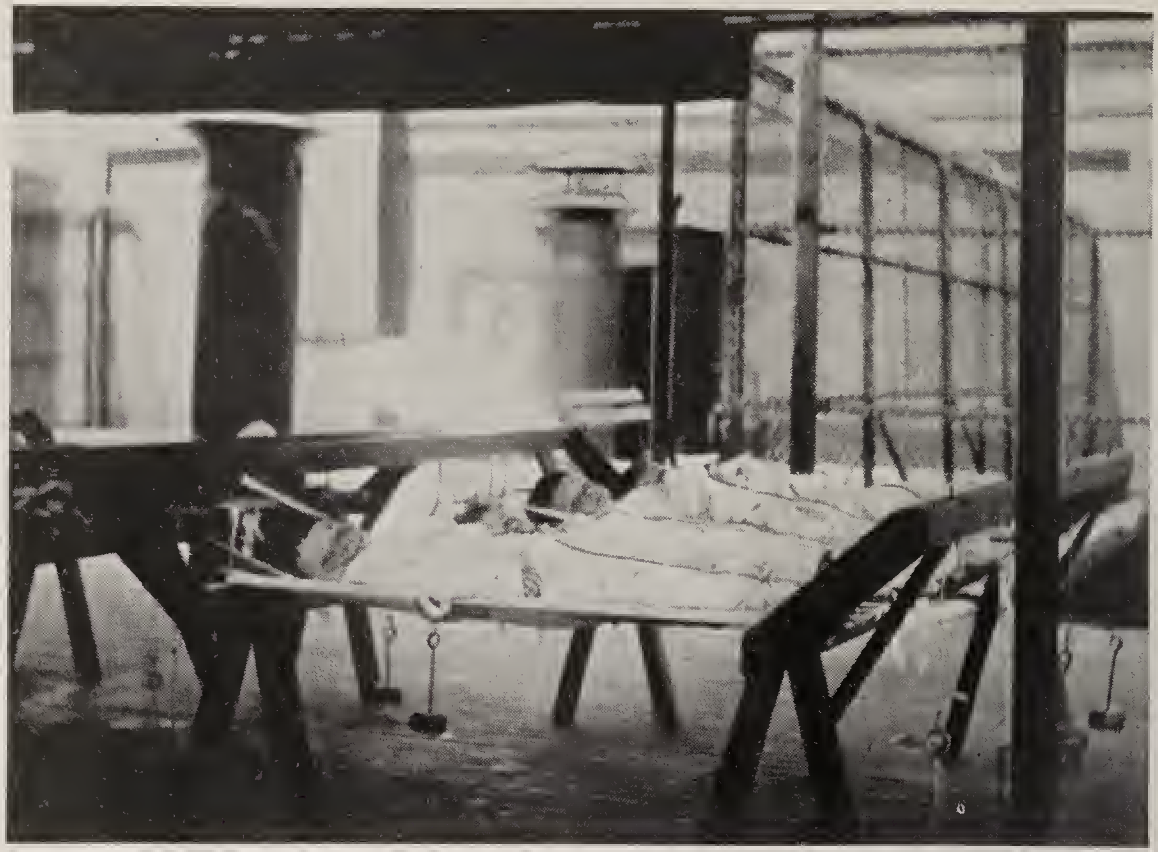


FIG. 58.—Roof garden with children on Bradford frames; head and foot extension in the cases of vertebral disease.

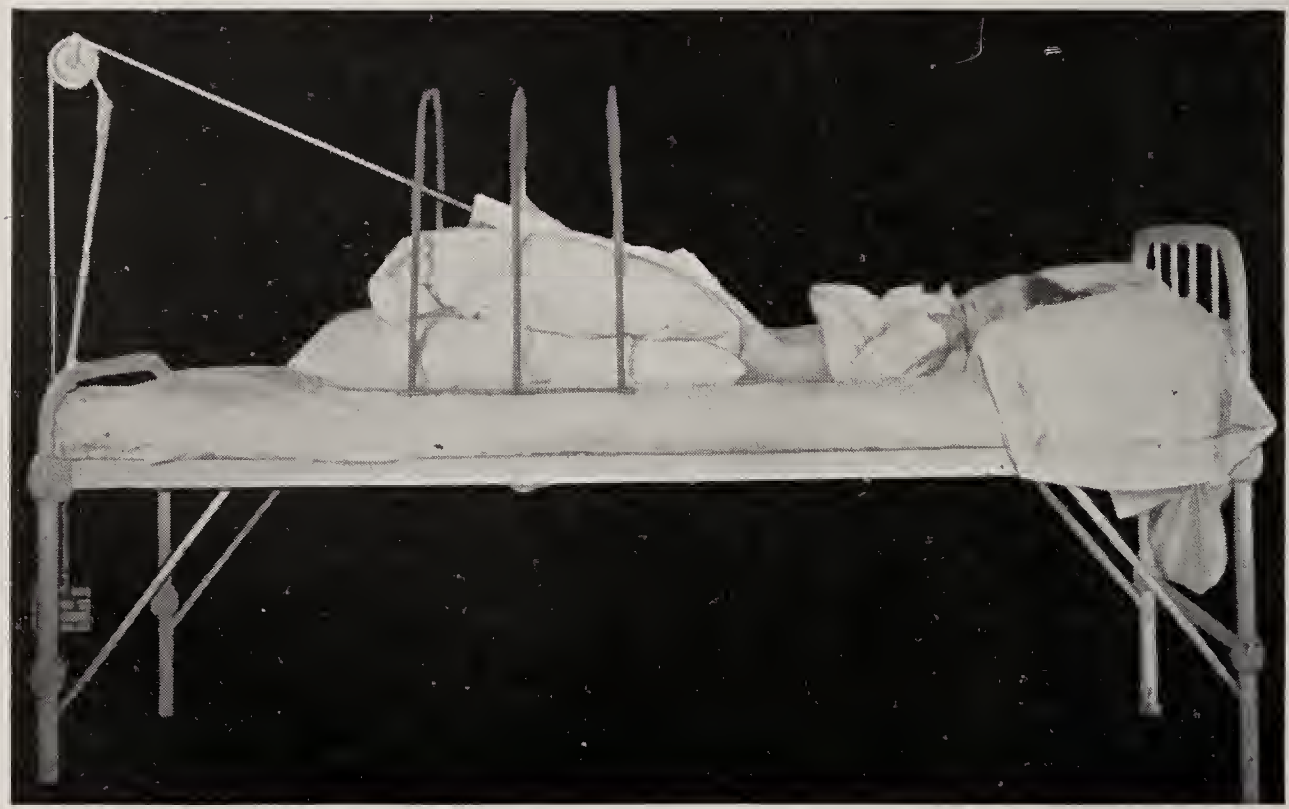


FIG. 59.—Tuberculosis of the hip with flexion deformity. The traction is applied in the axis of the deformity.

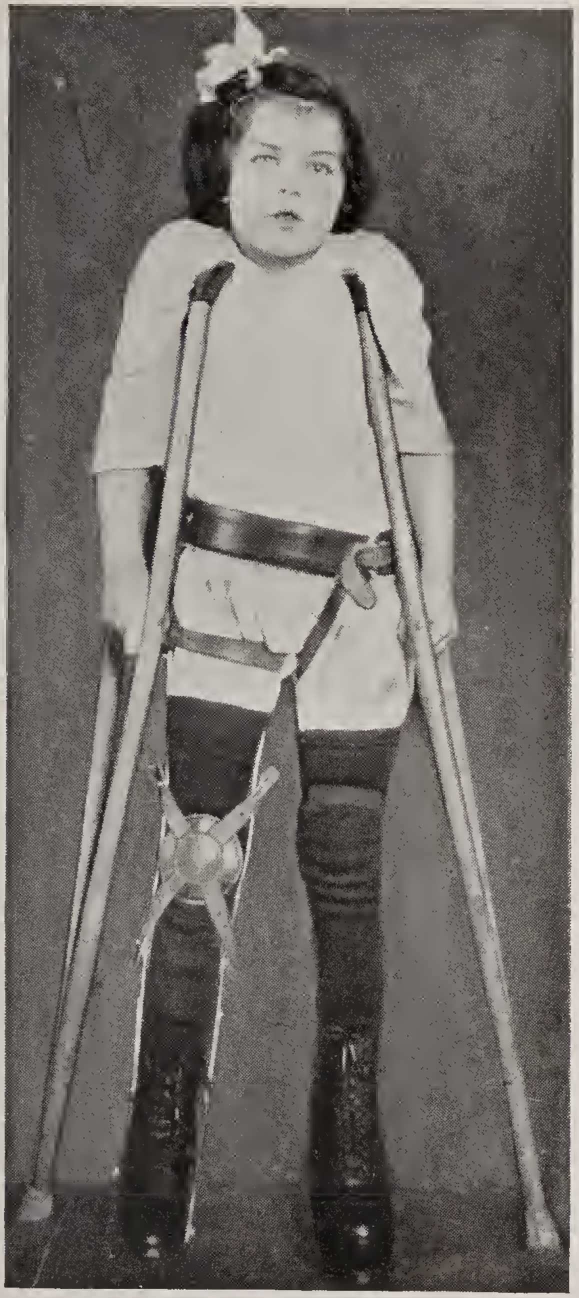


Fig. 60.—G. G. Davis's traction brace for tuberculosis of the hip. A high shoe is worn on the healthy (left) side, and the brace extends as a stirrup beneath the shoe of the diseased side; at each step the patient sits on a bar which passes beneath the tuberosity of the ischium, while the limb hangs free from pressure.

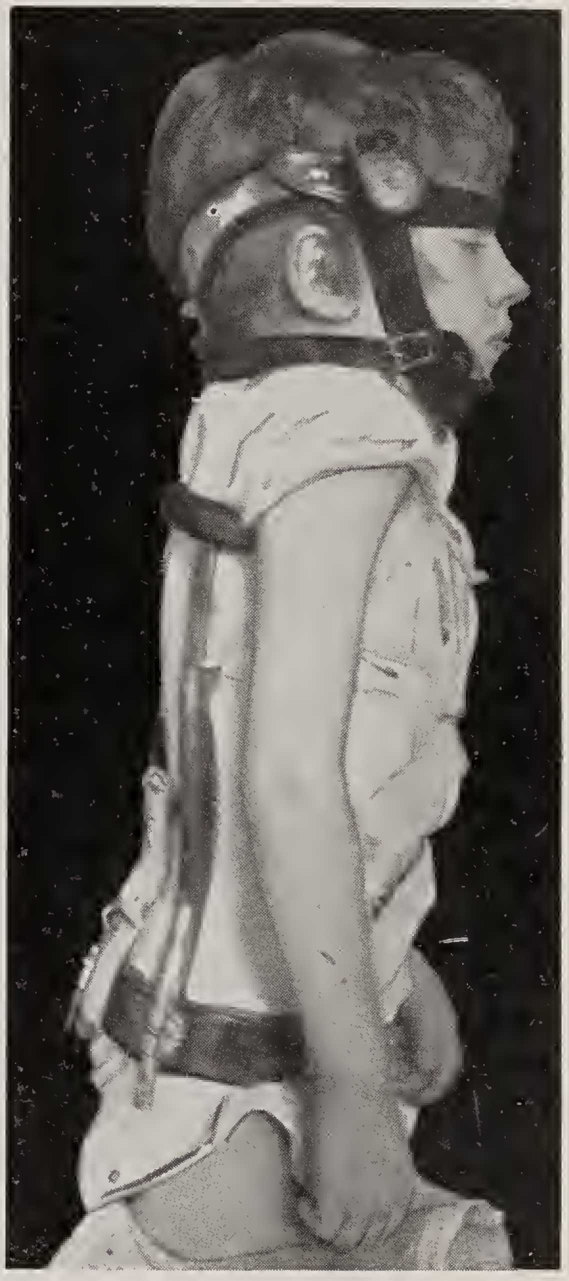


Fig. 61.—G. G. Davis's brace for tuberculosis of the upper thoracic or cervical spine. A band around the pelvis affords fixation below; the shoulders are supported by crutch-heads attached to side irons. A jury-mast supports a sling beneath the chin and around the forehead.

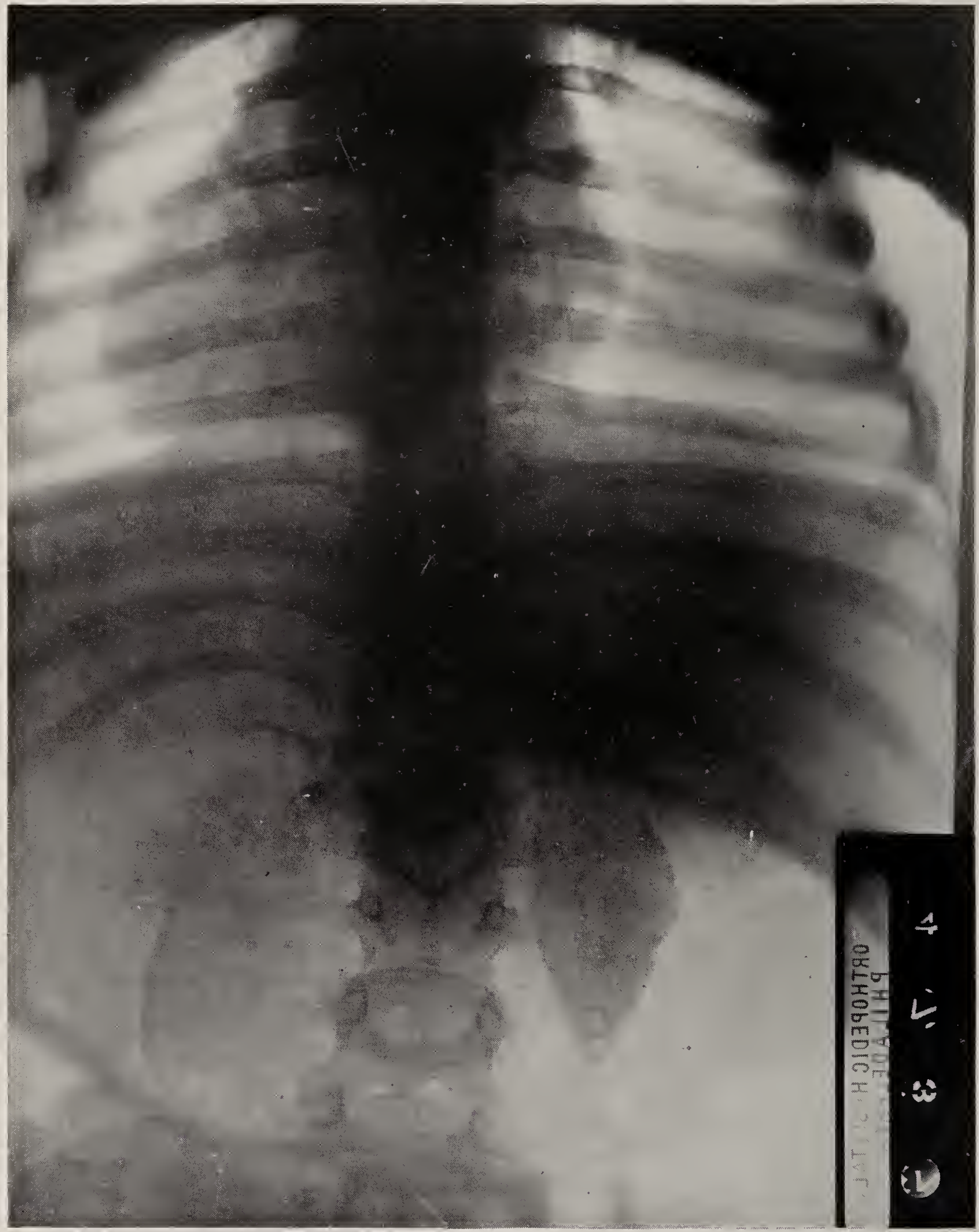


FIG. 62.—Bilateral psoas abscess (calcified) in an old case of tuberculosis of the lumbar vertebræ.

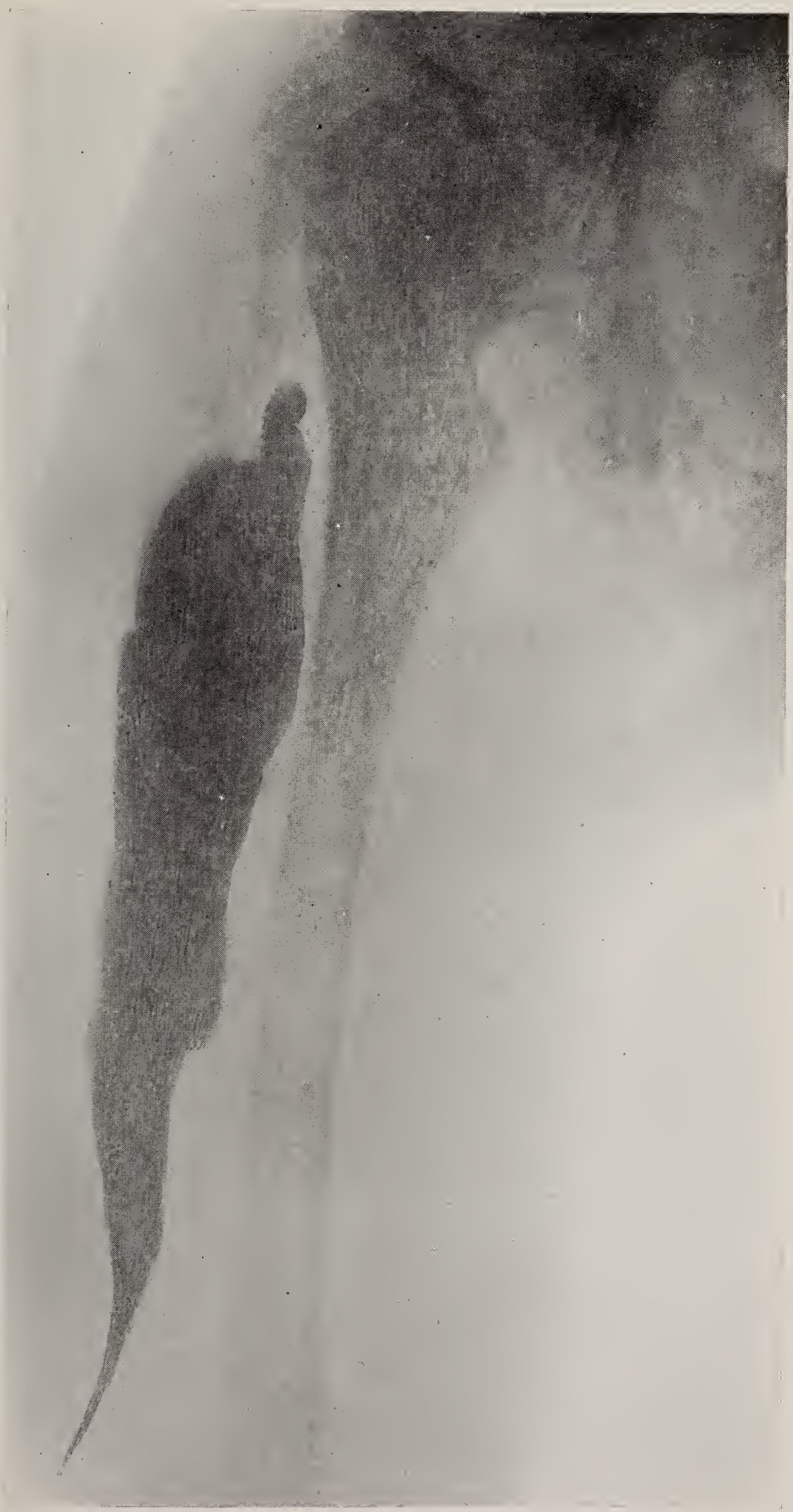


FIG. 63.—Sinus in a case of tuberculosis of the hip, injected from the opening on the outer surface of the thigh above the knee. Age 10 years. Sinus for 3 months; uneventful healing. In good health, with ankylosis of hip, 4 years later.

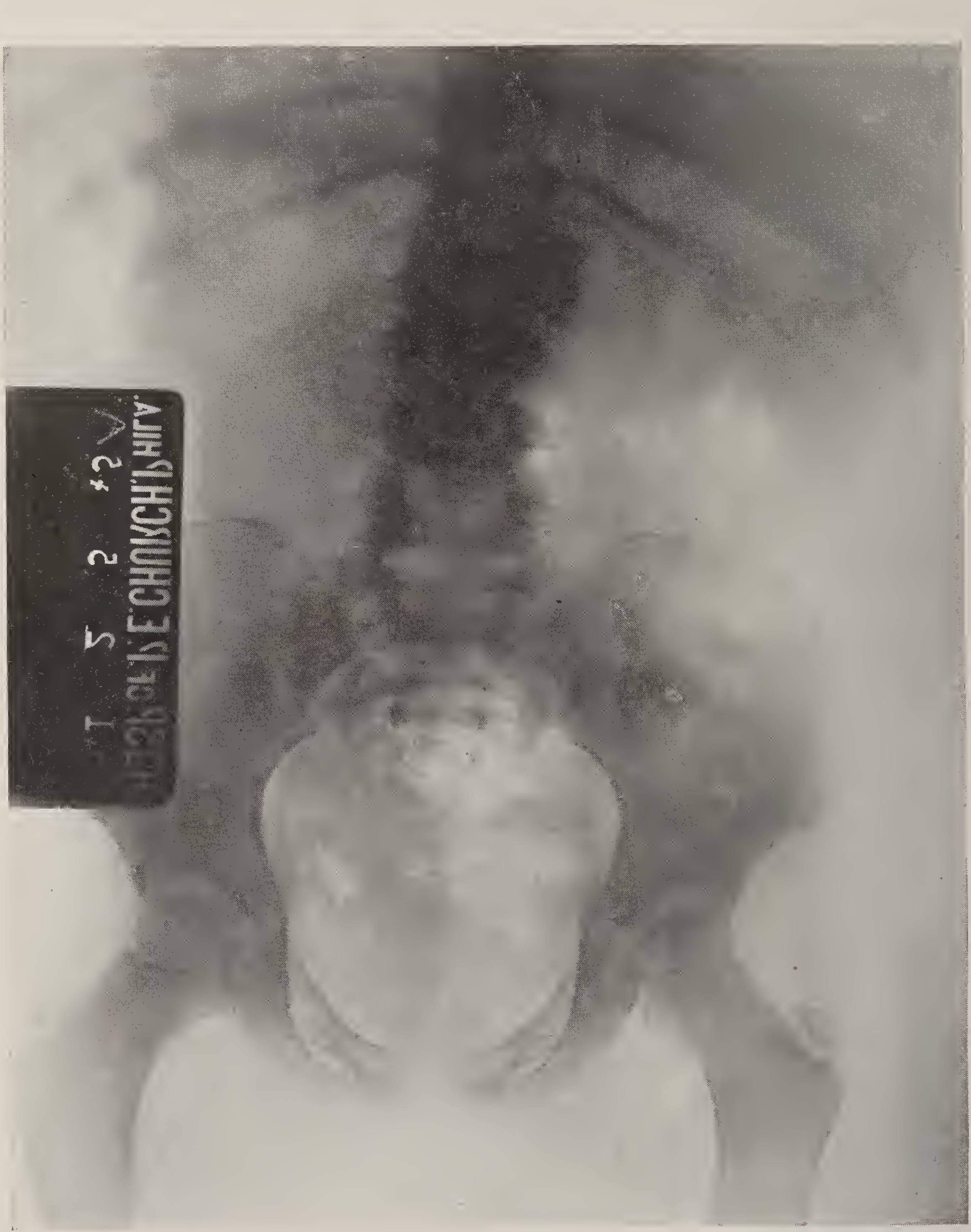


Fig. 64.—Tuberculosis of lumbar vertebræ. Age 4 years. (See Figs. 65, 66 and 67).

at a glance; but the older the child and the more calcareous matter there is therefore in his vertebral bodies, the more will the development of the kyphos be delayed. In adults the kyphos may not appear for many months after onset of symptoms. A recent kyphos is sharp and angular in outline; only after several years' duration does the hump become rounded. Seldom is there any occasion for doubt between a diagnosis of lateral curvature of the spine (scoliosis) and tuberculosis; but occasionally one sees a child in whom the tuberculous affection is confined to one side of a vertebral body, so that the whole vertebral column lists to one side. Such a *lateral deviation*, however, is not readily mistaken for a true lateral curvature. Have the child sit on the table, with hips flexed as far as possible, and with his body bent down between his thighs as far as possible. Then note whether the curve of his spine is one long normal convexity, or whether any area of it lacks the normal curve. Next turn the child prone on his abdomen, grasp both ankles and gently raise both high in the air, noting whether the spine preserves its normal range of hyperextension. Holding his lower body thus elevated by the feet, swing it gently from side to side, testing the lateral movements of the lumbar spine. Any lack of free movement in any region of the spine should make one suspect tuberculous disease. The mobility of the cervical and upper thoracic spine is to be tested similarly, by gentle hyperextension of the neck and shoulders. Examination should also be made for abscess complicating cases of tuberculosis of the spine, since this may appear before any kyphos becomes evident. Palpation in the iliocostal space may detect rigidity due to an early lumbar abscess long before bulging is apparent; and by the same means a psoas abscess often may be detected above Poupart's ligament before the pus has gravitated below this landmark, in the region on the outer side of the femoral vessels where it typically "points." Loss of hyperextension of the hip-joint, due to contracture of the psoas muscle, is an early and valuable sign: by bringing the child's pelvis to the edge of the table, as he lies on his back, and allowing both lower extremities to hang free, the contrast between the normal hyperextension of the hip on the healthy side and the persistent flexion on the side with psoas abscess, will at once become apparent.

Now, apart from the purely clinical diagnosis which has thus far been outlined, there are certain laboratory aids to diagnosis of which aid may be sought. The most important of these is of course the *x*-ray: this shows rarefaction and bone atrophy in the early stages; later also destruction and absorption of bone from weight bearing or pressure. In most cases the *x*-ray appearance is sufficiently characteristic, when the history and clinical examination are taken into consideration, for a positive diagnosis of tuberculous disease to be made; but there are cases where the diagnosis from other low-grade infections, from bone cyst, or even from sarcoma may not be very easy. And in the very early stages, where *x*-ray will reveal nothing that is characteristic, either because the disease is primarily in the soft tissues (synovia) of the joint, or because bone rarefaction and atrophy have not yet occurred—in the very early stages, I repeat, as well as in more advanced cases where a positive diagnosis seems difficult, resort to other laboratory examinations should not be neglected. The presence of syphilis usually may be excluded by a negative Wassermann test; and the von Pirquet test or even the hypodermic test by tuberculin (O. T.) may be helpful in proving the tuberculous nature of the malady. If another tuberculous focus can be found (lungs, lymph nodes), it is safe to conclude that the joint lesion also is tuberculous.

In cases where no satisfactory diagnosis can be reached the child should be treated as if suffering from tuberculosis, and kept under observation until the diagnosis can be settled.

3. TREATMENT. *Constitutional and hygienic treatment* are quite as important in surgical as in medical tuberculosis. The majority of patients with surgical tuberculosis are children of a school-going age. It is better for them to give up school for

one or two years, until their constitution is strong enough for them to conquer the disease than to attempt to keep up in their classes and grow physically worse and worse. You must not overlook the fact that it is better to have a healthy body with a stiff or deformed joint, than to have a straight and comely joint without a body capable of sustaining life. If the general health is good, joint function can be restored subsequently by operative means. It may not be possible for these patients to sleep in the open air, but they can at least sleep with all the windows in their rooms open, and be out of doors as much as possible during the day. In hospitals with suitable roof-gardens and sun-parlors it has been found that operative treatment is scarcely ever required. In institutions where for one reason or another it is impossible to keep bed-ridden patients out of doors constantly, it is at least possible for their beds to be wheeled out of doors and left out from morning until night. It is by no means necessary to have a hospital in the country for these patients: porches and balconies, even in the absence of roof-gardens will accomplish the same results in the most congested city districts.

Full, wholesome diet is equally important, and attention must be given to maintaining the appetite, regulation of the bowels, and such intercurrent affections as may arise.

Local Treatment may be summed up almost in one word: Rest.

Though the *modus operandi* of rest is not understood, a plausible theory is that suggested by Ely: he contends that cure is effected by abolishing joint function, because thus both red marrow and synovia become atrophic and in the case of ankylosis entirely disappear; and where these tissues do not exist, tubercle bacilli cannot live. Rest is obtained by *fixation* and *traction*. *Fixation* is secured by the use of splints, plaster cases, braces, etc., the sole object being to abolish motion at the diseased joint as effectually as possible. By *traction* is understood not so much actual extension on the limb sufficient to pull the joint surfaces apart, as cessation of weight-bearing and relief of pressure: in addition to relieving pain and securing rest, it also prevents deformation of the bone ends, which is prone to occur when weight bearing is continued. Traction is applied chiefly to the knee, hip and spine; fixation alone usually being sufficient in the upper extremity.

In all acute cases of tuberculosis of the spine and lower extremity the advantages of fixation and traction should be combined. This is best accomplished by bed treatment, so long as acute symptoms persist, regardless of the stage of the disease. In children the use of a Bradford frame, to which the body is strapped, provides fixation for spine, hip, or knee, in the most efficient manner. This frame is made of gaspipe, and is covered with tightly stretched canvas; it should be a little longer than the patient, and as wide as from one arm-pit to the other. The child is fastened to it by a broad canvas apron covering chest and abdomen, or by straps crossing the shoulders. The frame thus becomes a part of the child, and the two together can be carried about from room to room, or ward to roof-garden, thus preventing the painful and harmful joint movements necessitated by carrying the child in the arms or transferring it to a stretcher and back again to a bed.

Traction is applied by the ordinary Buck's extension apparatus, and *always in the axis of the deformity;* as muscle spasm lessens, gradual improvement in the limb's position may be secured.

When all symptoms of arthritis (limitation of motion from spasm, pain, fever, etc.) have been absent for a month or more, bed-treatment may be discontinued. This stage is reached in from two to six months in cases coming under treatment in the early stages of the disease. If, now, you recklessly discontinue local treatment (rest and fixation), in the erroneous idea that the joint is cured, and if the patient is allowed to resume joint function, it will be only a few weeks before symptoms of arthritis return, and possibly in aggravated form. It is absolutely imperative to guard the joint against injury and strain by continuing for a long period fixation or traction or both, during ambulatory treatment.

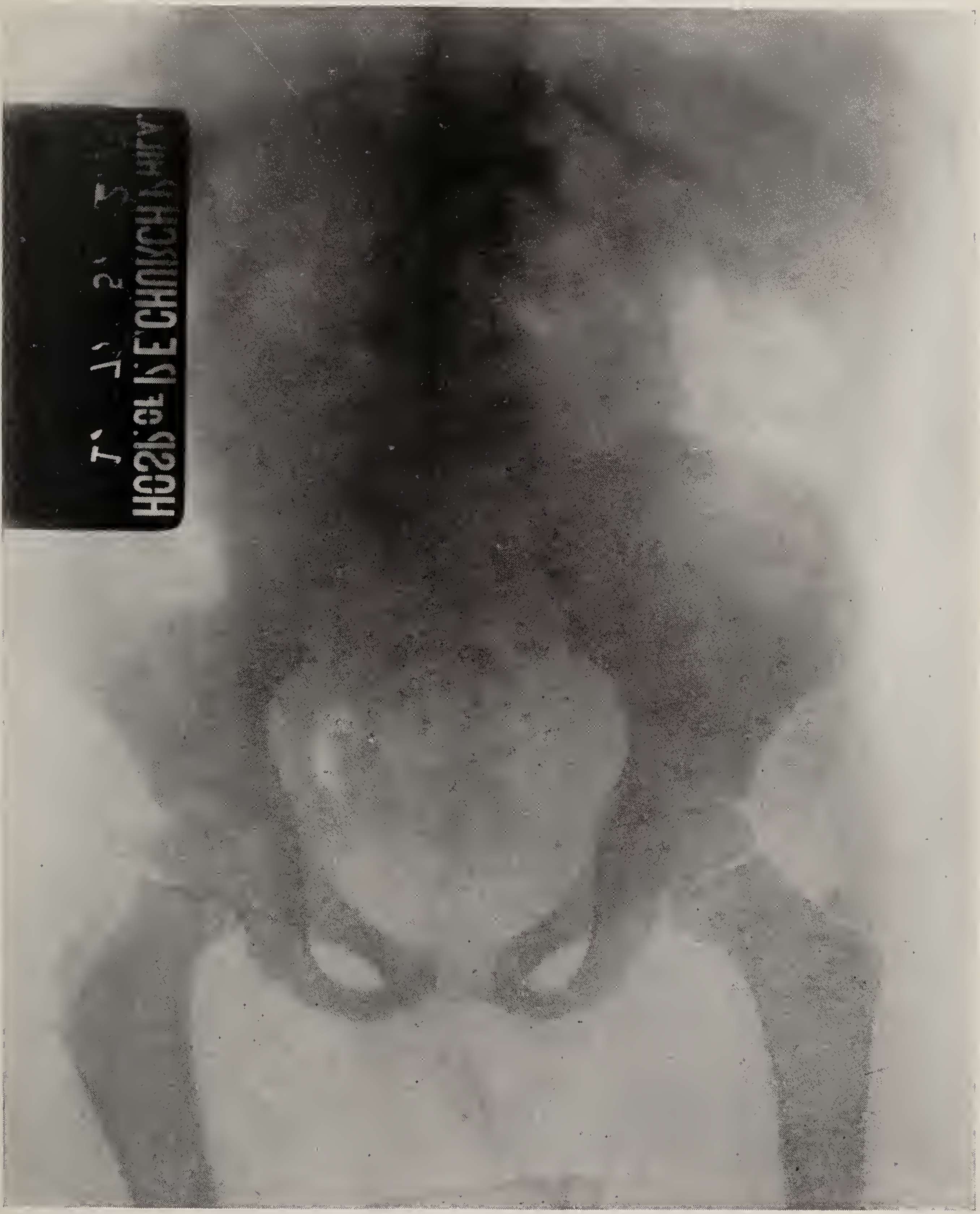


FIG. 65.—Same patient as in Figs. 64, 66 and 67, after bone transplantation.

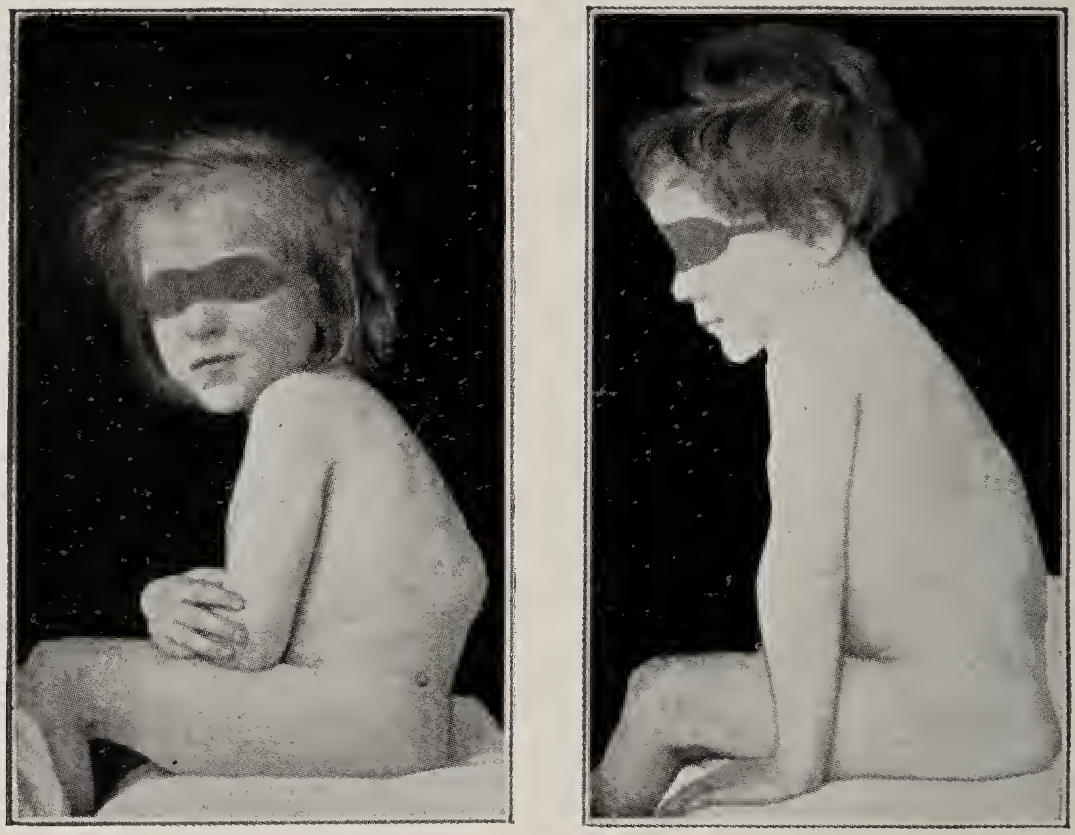


Fig. 66

Fig. 67

Figs. 66 and 67.—A girl, aged 4 years, with tuberculosis of lumbar spine (Figs. 64 and 65). Showing the hump, on admission; and the result of the bone transplantation after the deformity had been corrected by recumbent treatment.

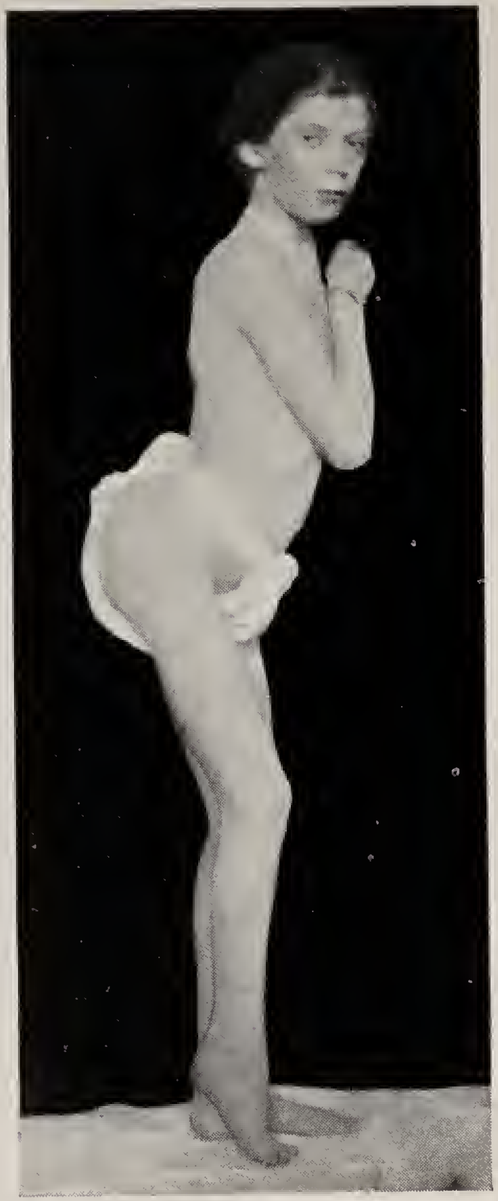


Fig. 68

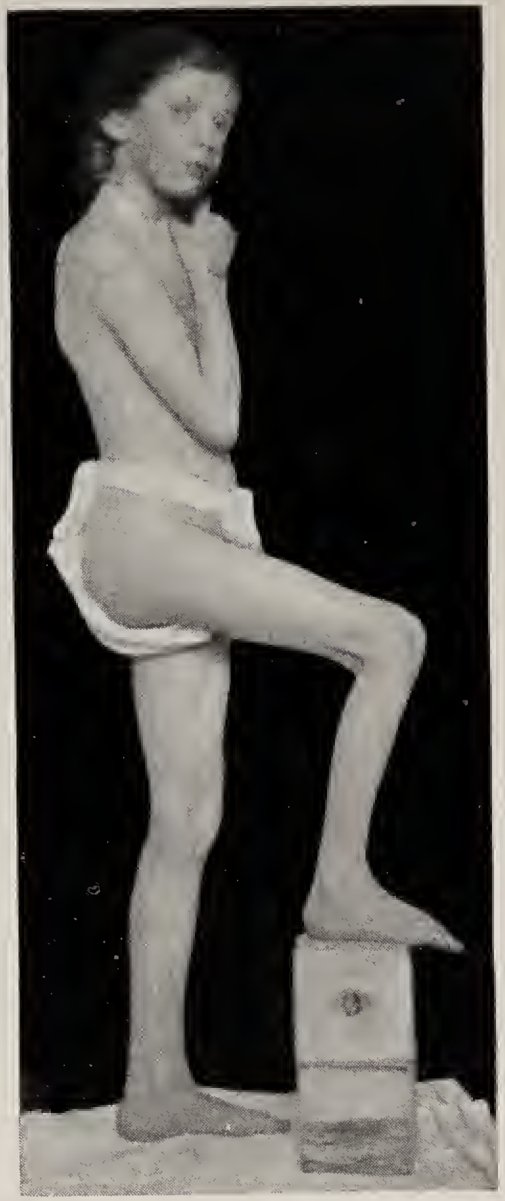


Fig. 69

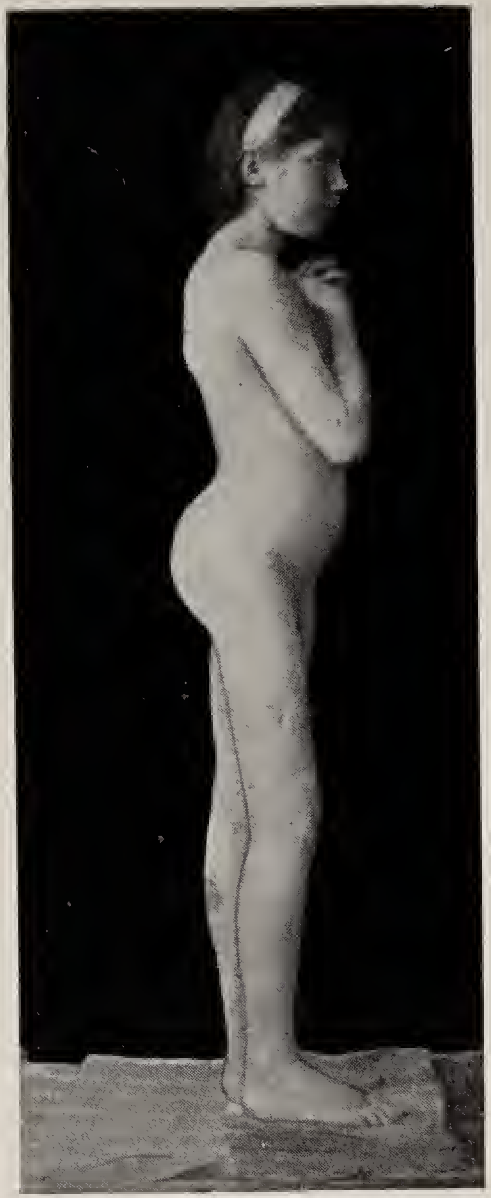


Fig. 70

Figs. 68, 69, 70.—Ankylosis of right hip in flexion deformity, the result of tuberculosis. Fig. 68 shows the extreme lordosis on attempts to bring the foot to the ground. Fig. 69 shows the lordosis corrected, but the hip nearly at a right angle. Fig. 70 shows the result of osteotomy of the femur below the trochanters. Though the hip is still ankylosed, the disability has been greatly lessened by correcting the deformity.

By the use of plaster-of-Paris braces and crutches both fixation and traction (in modified forms) can be continued; this should be done until, by allowing gradual return of function (first limited motion, then weight bearing), the surgeon proves that the joint lesion has become so thoroughly encapsulated as not to be liable to cause recrudescence of the disease.

This period of ambulatory after-treatment extends always through months, sometimes for years. Only by making haste slowly can permanent good results be achieved.

Treatment of Cold Abscess. If the joint be put at rest and the patient kept in the open air the threatening abscess may cease to enlarge and may gradually disappear. If in spite of these conservative measures the abscess continues to enlarge and threatens to approach the skin, with the consequent danger of infection from the skin cocci, even before rupture makes such an infection sure, you will find it safer to expose the abscess wall by careful dissection through the overlying healthy tissues; swab these with 3 per cent. iodin, evacuate the contents of the abscess and wipe its cavity dry with iodoform gauze. The incision to reach the abscess is then sutured in layers without drainage. Often the operation may be done under a local anesthetic. In many cases the incision heals without breaking down at any point, and in only a few does the abscess refill and require a second evacuation. Should the incision break down it will nevertheless usually heal by granulation. It is dangerous to leave a cold abscess alone until the overlying skin has become adherent and reddened, since secondary infection is then very frequent, and rapid joint disintegration, hectic, amyloid disease, etc., follow; and it is still more dangerous to open a cold abscess without perfectly aseptic technic, or to drain it by tube or gauze after incision, or to allow it to discharge itself spontaneously.

But sometimes the patient is not seen until spontaneous rupture threatens and secondary infection is already present.

Then the abscess should be evacuated by a puncture where it is pointing, but should not be drained; if the puncture is occluded by aseptic gauze, in many cases little or no subsequent discharge will occur, the "hot" will gradually resume its character of "cold" abscess, and eventually may be absorbed. Thus the formation of sinuses and prolonged suppuration may be prevented.

Aspiration of a cold abscess I believe is inferior to formal incision because it cannot be done satisfactorily until the pus is very close to the surface and unless it is very fluid.

Treatment of Sinuses. Sinuses nearly always indicate secondary infection. If no secondary infection is present (a fact which cultures of the pus will demonstrate) they usually will heal under rest and constitutional treatment. Exposure to the sun's rays or on cloudy days to artificial light encourages healing of tuberculous sinuses. Incandescent bulbs may be suspended from a cradle over the patient's bed, and nearly constant exposure thus be provided for. Direct sunlight must be used cautiously at first to prevent sunburn. If the sinuses fail to heal and the discharge of pus is not profuse, they should be filled with Beck's bismuth paste: one part of bismuth subnitrate (arsenic free) to two parts of sterile amber vaselin. This is heated on a water-bath until fluid, and is injected under pressure into the sinuses by a glass syringe, which after being boiled is rinsed in alcohol and allowed to become thoroughly dry before it is filled with the liquid paste. Skiagraphs made after the injection will demonstrate the origin and ramifications of the sinuses. If the sinuses are very large it will be safer to use only a 10 per cent. bismuth paste instead of Beck's original formula, for fear of bismuth poisoning.

If profuse suppuration persists in spite of conservative measures, and particularly in cases of long-continued hectic, the sinuses should be opened freely and any sequestra should be removed. Such sequestra, though usually very small, as well as carious bone, form favorable sites for the development of saprophytic bacteria, and if the pabulum of the latter can be removed, the hectic fever may be checked.

Operative Treatment. In children it is only in the class of cases just mentioned that operations, such as excision of joints, are required. But in cases of tuberculosis of the vertebræ, bone transplantation may well be adopted for the purpose of providing internal fixation and thus arresting the disease. But under the age of six years I believe it is usually inadvisable, as being neither positively indicated nor so certain in its results as in older children and adults.

Treatment of Deformities Following Tuberculous Arthritis. Many patients who have been inadequately treated, and some in spite of the best treatment, develop in the course of years certain deformities which also are positive disabilities. For these it is possible to do much by operative surgery. But one should always bear in mind that there is a possibility of lighting up again the old infection, and should consider well whether in the first place the patient's disability is so pronounced as to demand relief, and also whether the means of relief proposed are reasonably certain and adequate. Mere stiffness of a joint cannot be considered an indication for operation so long as it entails no particular disability. Ankylosis is the best and surest cure of tuberculous arthritis, and except in the elbow or shoulder usually should not be interfered with. In both shoulder and elbow, particularly the latter, excision gives admirable results with free motion. But in the lower extremity, unless the ankylosis is in bad position, it should not be disturbed; attempts to restore motion are not likely to be very successful, and stability rather than movement is desirable in the hip and knee. Ankylosis at the hip in bad position may be successfully treated by subtrochanteric osteotomy of the femur, the position of the limb being improved, but ankylosis being preserved. Deformity without ankylosis at the hip may rarely be improved by cautious correction under an anesthetic, but usually, and especially is this so if there is pathologic dislocation of the hip; an open operation will be required, with replacement of the end of the femur in the acetabulum an effort being made to convert the flail and helpless limb into one capable of supporting the patient's weight.

So one can see that the "cure" of tuberculous bone and joint disease is a long and tedious process; that the motto *principiis obsta*—resist the beginning of the disease—is true especially of this form of surgical disease; and that the only way in which treatment can be adopted early enough to be really efficient, is by making the diagnosis without unnecessary delay. I have tried, therefore, to explain to you how the diagnosis usually may be made merely by thorough and systematic physical examination, and I hope you will bear in mind that it is your duty as pediatrists to recognize these cases, and if you are not surgeons yourselves, with mechanical tastes and with proper facilities at your command, I beg you to refer your patients with tuberculous bone and joint disease to hospital services where proper treatment may be promptly instituted and conscientiously continued as long as may be necessary.

THE RELATION OF THE CRIPPLE TO THE PUBLIC[1]

By A. BRUCE GILL, M.D.
ORTHOPEDIC SURGEON TO THE HOSPITAL

In these few remarks I desire to present a broad view of the subject of the relation of the cripple to the public and of the public to the cripple, and to point out the necessity of our formulating some comprehensive plan along which we should work toward the attainment of the ideal end. If this ideal end seems too far distant or too lofty, let us not forget that the world is moving rapidly in these latter times and that the dream of today may have become past history by the day after tomorrow.

The cripple is a defective. He lies outside the wall of the normal—I say *wall* purposely. There are other defectives, the mental and the moral, who also lie outside the pale. For ages what has been the attitude of society and the state, *i. e.*, of the normals within the wall, toward all defectives who live without? They have shut their eyes that they might not see; for the abnormal is a deformity, an ugliness, that shocks the sense of the beautiful and disturbs one's mental and emotional placidity. This attitude persists today in many people, who shun the sight of cripples because it "makes them feel bad." Organized society has recognized defectives only as they have been a menace to society. They have been made paupers, beggars, and criminals; and have been segregated in almshouses, in asylums, and in prisons. They have been starved, punished, and killed. Like the lepers of old they have been driven without the city, beyond the life and the walk of the clean. And it has been left almost alone to occasional private charity to go forth to seek the defectives, whether physical or mental or moral, and to take them by the hand as a brother or sister, to cleanse them, to cure their deformity, and to lead them back into the city beautiful.

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[1] Read by invitation before the Section on Industrial Medicine of the College of Physicians of Philadelphia on October 17, 1917.

How long has it been since the criminal has been treated otherwise than a vicious man who must be punished or killed? How long has it been, even, since our prisons and almshouses and asylums have been made cheerful and comfortable and home-like places to live, rather than dull gloomy dens almost comparable to the tombs in which the lepers dwelt? How long have these unfortunates been decently clothed and fed? Stripes and hideous uniforms have not yet disappeared. What serious effort has organized society ever made to cure defectives as far as possible, to train them to occupy a useful place in the world, and to admit them to our daily walk in life whether of business or of pleasure? Is it easy for a man who has spent a term in a penal institution to find honest employment after his discharge? And what opportunity has the cripple to learn to do a man's work and to find admission to the usual occupations?

But a great principle is slowly working to alter the old relation of the normal to the abnormal, to open our eyes, to remove the disgust and intolerance toward the weak, the deformed, and the unfortunate. The truth is working in a hundred different ways which we see but dimly or not at all. Prison reforms of all sorts, the various investigations into the causes and conditions of the social evil and other vices, the establishment of homes and schools for the care and training of cripples and other physical defectives, the founding of societies to find honest employment for various kinds of unfortunates—all these and many other recent activities are but manifestations of the truth that it is the serious duty of society to recognize that the abnormal does exist, that it is a deadly menace to the welfare of mankind, and that it is to be treated not by expulsions or by punishments or by spasmodic charity, but by comprehensive systematic training and development that the abnormal may grow more like the normal and that the defective may become an efficient member of society.

We have long recognized the advantage of training and developing the normal individual to the utmost. Schools, public and private, colleges, institutions of a hundred kinds have for centuries been training the youth of the world; and our present wonderful era of knowledge, science, prosperity, and comfort is the result of this prolonged effort. Has not the time come for the systematic care and development of that large body of defectives who are a menace as well as loss to society? It is the idle, the poverty-stricken, the weak-minded, the crippled, who constitute the vast underworld that has always been a deadly menace to civilized society and that is ever ready at the slightest opportunity to swarm from cellars, sewers, and slums with hand raised against every man. The normal has been tended and reared and has grown into the great tree of civilization; but there are countless creeping, crawling things that are gnawing at its roots and digging into its sap-wood. It has been said that the marvelous civilization of the ancient Greeks was only the illuminated top of a vast pyramid whose base lay in shadows. The few were highly educated and wealthy, the many were paupers and slaves. The safety of our civilization lies in the education and enlightenment of all classes of citizens.

Now arises the question as to how this shall be accomplished. We have but to open our eyes to the marked trend of the present times, and our question is answered. It is a truism that the whole is composed of its parts. From this it follows that the people as a whole, *i. e.*, organized society, whether the community, the state, or the nation, should concern themselves with the welfare and the development of the individual, and that the individual or groups of individuals should be controlled for the welfare of the whole. Centralization of power, socialism—call it what you will.

We are not fighting centralization of power that is lodged in the German Government, for Wilson and Lloyd George probably hold in their hands much greater authority than does the German Kaiser; nor shall we soon forget the lesson we have learned of German organization and efficiency. We cannot deny it, the great tendency of the times is toward central control of the great industries and activities of national life. But this control shall be for the benefit of the people, and the heads of power shall be answerable to the democratic whole, not to the autocratic few.

Nor shall this power be limited to the control of railroads, coal and copper mines, water supplies, forests, the bread we eat—the physical things vital to the welfare of all. It governs as well the education of our children, and the health and comfort of the people. Are we not only beginning to realize that the strength and health of our citizens vitally concern the nation. They are our greatest assets, both in times of peace and in times of war. For years far-seeing physicians have demanded that a health officer should sit in the cabinet at Washington. Now in the time of crisis the ablest members of our profession are summoned to the nation's need. Organized society should provide for the proper care and maintenance of the poor, the sick, and the cripple who cannot care for themselves. Our present methods are a great injustice to the medical profession which takes largely upon its own shoulders the care of the sick who are too poor to pay for proper attention. Our hospitals are maintained by private charity and by public purse, but the surgeon and the physician receive no recompense for their long hours of labor there. That this principle of central control of health is making headway is shown in the growing power of officers and boards of public health, in the medical care given to children, enactment of laws for the compensation of industrial accidents, in the establishment of state institutions for the care and education of cripples, and in the efforts made to segregate the feeble-minded and the insane. But only the beginning has been made.

Look forward through the years and see with the mind's eye a reconstructed world. There will be no idle and no poor, for there will be no rum and every man and every woman shall have a work in the world for which he has been trained and for which he shall receive just compensation. The things that drive men to crime shall be no more. And the weak and the feeble-minded, who constitute so large a portion of the criminal class, also shall have a work to do so far as they are able, or shall, at least, lead a happy and peaceful life where they may no longer menace society by their actions or by the propagation of their kind.

Following this great war there will be a vast reconstruction of ruined cities and lands, of crippled men, of governments, and of the lives of the people. The world will never again be what it was before. The times are rushing forward incredibly fast. In the new life the poor shall hold up his head, and the cripple shall walk and have an honorable place among men.

In conclusion it may be said that while the cripple is physically handicapped, in other respects, he is, in most cases, a normal individual. Naturally he is as full of energy and ambition as are other people, and desires to occupy a useful and independent position in the world. But he needs a chance. And the public owes it to him and to itself to give him this chance, to afford him at least equal opportunity with others to secure an education and to fit himself for work and to secure employment for which he is suited.

This, I take it, is the proper relation of the cripple to the public and of the public to the cripple. When this war shall pour into our midst thousands of crippled men, then the issue shall be forced upon us and we shall be obliged to enter upon some comprehensive system for their care.

BIRTH INJURIES OF THE SHOULDER[1]

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THE clinical entity known as brachial birth palsy has been the subject of a number of rather controversial papers in recent years. These controversies have had to do both with the pathogenesis of the affection and with its treatment. The present contribution to this discussion is based on a careful study of about forty patients seen within the last few years in my services at the Episcopal and Orthopædic Hospitals, and is an effort to systematize the treatment of a condition still too frequently overlooked by the average practitioner, or, even if not overlooked, too often neglected. It is difficult to systematize the treatment of an affection whose pathogenesis is not known; but in spite of much study I must confess that my views on the latter subject are still uncrystallized. But I know that much good may be accomplished by definite lines of treatment and much harm done by neglect or by misdirected efforts. This is, in fact, one of those departments of surgery in which Science still lags behind Art; but as experience accumulates there is every reason to believe that Art, like Wisdom, will be justified of her children.

First brought prominently before the profession in 1872 by Duchenne, it was an affection long regarded as of interest solely to neurologists, owing to the prevalent theory as to its causation: Duchenne taught that the lesion was in the brachial plexus, but as his observations are recorded in a text-book devoted to Local Electric Treatment he did not enter into the question of exact anatomical localization of the lesions. Erb, in 1874, studied the similar but much rarer affection in adults, and specified more particularly that the lesion occurred at the junction of the fifth and sixth cervical roots of the brachial plexus. He thought it was due to direct pressure at this point ("Erb's point"). The teachings of Duchenne and Erb have dominated the minds of medical men until within recent years. It is true that Küstner, in 1889, stated that all such cases seen by him really were instances of separation of the upper epiphysis of the humerus; but his views were not accepted by others. Moreover, Whitman, in 1905, in a brief paper which seems to have been completely ignored until recent years, called particular attention to posterior dislocations of the shoulder often associated with this condition, and urged rational treatment of the dislocation when present on the plan adopted in cases of congenital dislocation of the hip. He expressed the belief that true congenital dislocation of the shoulder was exceedingly rare, as was also a dislocation due to injury during birth; holding that the posterior dislocations so often accompanying brachial birth palsy were the result of the paralysis, not of the birth injury. A. S. Taylor, still upholding the purely neurogenous theory, reported in 1907 a series of cases in which operation had been done by him on the brachial plexus, and referred to some experiments by himself (1905) tending to show that it was possible for the roots of the plexus to be ruptured by traumatism (see paper by Clark, Taylor and Prout).

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[1] Read by title before the Philadelphia Academy of Surgery, October 1, 1917. Reprinted from Annals of Surgery, 1918, i, 25.

Meanwhile, general surgeons had been studying cases of the similar lesions seen in adults, to which Erb at first called special attention. Duval and Quillain, in 1898, as a result of their studies, came to the conclusion that there were no such clinical entities as paralyses due to lesions of the brachial plexus, only two types existing, *radicular* and *terminal*, affecting either the spinal motor roots or the nerve-trunks below the plexus. Subsequent observations appear to have confirmed these conclusions, both as regards the injuries occurring at birth and those encountered during adult life. Delbet and Cauchoix in 1910 collected 36 cases of paralyses complicating dislocations of the shoulder: 25 of these were *terminal* paralyses, and were the sole lesions produced by the dislocation itself; the remaining 11 lesions all were *radicular* and were due not to the dislocation, but to the cause which produced the dislocation. There were no true lesions of the brachial plexus in this series.

Now T. Turner Thomas, who had been studying the "stiff and painful shoulders" for which Codman and others have erected a pathology based on so-called subdeltoid bursitis, turned his attention to the pseudoparalyses often, if not indeed usually, present in these cases, and at a meeting of the Philadelphia Academy of Surgery in October, 1910, proposed the theory, to which he has steadfastly adhered, that in almost all such cases, birth injuries or adult injuries, the supposed paralyses are secondary to a primary lesion of the capsule of the shoulder-joint; that the effused blood, lymph and synovial fluid catch the nerves in cicatricial tissue; and that the dislocation so frequently accompanying the birth injuries is primary, and therefore the cause, not the result, of the brachial palsy. Similar teachings were subsequently (June, 1912) promulgated by Lange; but neither Lange nor Thomas has been able to secure general recognition of the truth of their doctrines.

Sever and J. J. Thomas (1916) have recently published two studies based on 470 cases of brachial birth palsy, personally observed by one or both of them. Sever refers to his "numerous dissections on infantile cadavers" which showed that "traction and forcible separation of the head and shoulder puts the upper cords, the fifth and sixth cervical roots of the brachial plexus, under dangerous tension." This confirms Taylor's experiments of eleven years previously, and it may perhaps be accepted as proved that such a lesion can occur during obstetrical delivery by a similar mechanism.[1] The theory of direct pressure on the plexus as a cause has been abandoned. In his experiments Sever was unable to rupture the joint capsule, to separate the epiphysis, or to dislocate the head of the humerus. He notes, however, that in 1900 Stone had been able easily to separate the epiphysis; and T. T. Thomas says (*loc. cit.*, 1914, p. 212) that he has in several infant cadavers obtained by hyperabduction an epiphysial separation or a fracture of the upper end of the humerus, but the capsule was stronger than the humerus, as suggested by Lange. But the contention of Thomas is that rupture of the capsule is produced not by hyperabduction (which, as already noted, produces epiphysial separation or fracture) but by direct pressure on the humerus and acromion by the maternal pelvis, the arms being flexed and adducted at the shoulder (*loc. cit.*, 1914, p. 212).

As a result of these divergent views of the pathogenesis of the condition, there has been equally great divergence in the methods of treatment recommended; and until within the past ten years no very active treatment of any kind has been instituted even by neurological or orthopedic surgeons. My own active interest in the condition was aroused not much more than five years ago, by the apparently increased frequency with which patients of this type were being seen, and the consequent necessity of systematizing their treatment. My views of the pathogenesis were briefly expressed in discussing the views of Thomas already noted (*Tr. Philadelphia Acad. Surg.*, 1914, xvi, 235; *Annals of Surgery*, 1914, lix, 142). These views I still hold:

"First, that pure nerve lesions occur, and may be of much greater importance than any injury to the shoulder-joint even if this is present; and, second, that posterior subluxation of the humerus is a frequent lesion, often overlooked, and perhaps may be the cause of persistence of paralysis." Dr. Thomas, in reply, said "that he did not mean to say that none of these cases of birth palsy were due to rupture of the brachial plexus, but he believed that none of his twelve cases were. He thought it fair to say that most cases are not."

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[1] However, it may well be, as suggested in a personal communication by T. T. Thomas, that the preservative fluid makes the brachial plexus of infantile cadavers more prone to laceration than is the plexus in the normal baby at birth.

Formerly, babies with "birth palsy" which came under my notice at the Orthopædic Hospital, in the services of my chiefs, G. G. Davis and R. H. Harte, usually were referred to the Nervous Department of the Hospital, and seldom or never returned for observation by the surgeons. I remember asking the neurologists what became of such patients in adult life; and the consensus of opinion seemed to be that they all recovered without disability, as none of the neurologists whom I questioned had ever seen an adult who had had "birth palsy." I remember also that occasionally when these babies were brought to us at a very early age and the shoulder seemed particularly painful, the diagnosis made was epiphysial separation of the upper end of the humerus; then they were retained under surgical care, the arm bandaged to the side or in a sling, until acute symptoms subsided, and, after being referred to the mechanotherapeutic department for massage, were again lost to sight. I remember only one case diagnosed as "congenital dislocation of the shoulder"; this was in a boy of seven years or thereabout, with a subspinous dislocation of the left shoulder, and with the history that it had been present from birth. There was no question of the presence of the dislocation; and though the question of the etiology (whether truly a congenital deformity, or from trauma during parturition) was discussed, no satisfactory conclusion was reached, and as no active treatment was urged, the patient did not return. There was only slight disability and no apparent paralysis.

When I began to pay particular attention to these patients, stimulated largely by conversations with G. G. Davis and T. T. Thomas, it did not take very long to learn that the reason they had ceased to be brought to the clinic was not because no disability remained (though this was the case in a few) but because the parents were led to believe the condition was hopeless and that nothing more could be done.

It is the contention of the neurologists that the lesion occurs in most cases at the junction of the fifth and sixth cervical roots as they join to form the upper cord of the brachial plexus. This point is just above the origin of the suprascapular nerve supplying the external rotators of the shoulder; and it is these muscles which are most constantly paralyzed. They point out, moreover, that from the fifth and sixth roots comes also the musculocutaneous nerve, supplying the flexors of the elbow (biceps and brachialis anticus); while the axillary (circumflex) nerve supplying the deltoid (usually also paralyzed) comes almost entirely from the fifth cervical. But they conveniently ignore, or seek to explain by plausible arguments, contrary facts, such as that the very same nerve roots transmit fibers for the subscapularis muscle, the pectoralis major, the latissimus dorsi, the teres major, and the pronator radii teres muscles none of which, as a rule, exhibit any lasting paralysis, if indeed they were not intact from the very moment of the injury. A. S. Taylor, Fairbank, Sever, and others, all acknowledge the truth of the common teaching that the subscapularis is supplied by fibers derived solely from the fifth and sixth cervical, and yet it is never found paralyzed. Fairbank alone boldly faces the dilemma, and, though he admits freely that this muscle by its contraction is responsible for the posterior subluxation of the shoulder, being unopposed by the paralyzed external rotators, puts forth this explanation: that "recovery of the nerves has taken place before this deformity has occurred or at any rate become fixed, and in cases in which the paralysis is permanent the subluxation does not occur." This is clearly an untenable theory; if recovery of the nerves to the subscapularis occurs with such regularity, why does not recovery of the nerves to the other muscles also occur, if the lesion has been where all the fibers pass in a common trunk? Moreover it is simply not true that posterior luxation does not occur when the paralysis is permanent; witness my Case V. Unless indeed, he means that posterior luxation does not occur if the subscapularis remains paralyzed; but I do not know of any cases reported in which paralysis of this muscle has been observed. A. S. Taylor seems to have had but a confused idea of the effect of these paralyses: he refers to the deformity which develops from *contractures of the paralyzed muscles and the ligaments of the joints.* Thus he assumes that *contracture* of the subscapularis occurs because it was paralyzed (of which there is no evidence), but he attributes *relaxation* of the external rotator muscles to their paralysis, and yet considers the limitation of elbow extension due to *contractures* of the paralyzed flexor muscles. It is despair over a theory such as this, which blows hot and cold out of the same mouth, that encourages others than neurological surgeons to look elsewhere for an explanation of the phenomena encountered in this still obscure condition.

Unless the anatomy commonly known is incorrect, I do not see how the immunity of certain muscles, especially of the subscapularis and teres major, can be explained, if the nerve lesion is supposed to be at the juncture of the fifth and sixth roots. The descriptions of what has been found at operation soon after birth are so vague that not much reliance can be placed on such observations; this vagueness probably is due to the impossibility of telling, even with the nerves exposed to view, how much they were damaged. In cases operated on months or years after the injury occurred, the extent of the scar tissue and the difficulty of the dissections render such observations also of limited value. It may well be, as indicated by some of these operations, that the suprascapular nerve itself (below the plexus) is damaged at the time of the original injury. Indeed it has occurred to me that it may be not improbable that all the nerve lesions in the majority of these cases are *terminal* and not *radicular*. Consider the course of the nerve-trunks (terminal) which supply the various groups of muscles: *all the muscles most constantly paralyzed are supplied by nerves which pass very close to the shoulder-joint*[1] and *ipso facto* are liable to injury; whereas *the muscles which habitually escape paralysis are supplied by nerves which at no part of their course come into close relation with the shoulder-joint or the bones which compose it.*[2]

This seems to bring us very close to the theory of Thomas and Lange, that the primary lesion is in the shoulder-joint, and that involvement of the nerves occurs secondarily. It is indeed a question in my own mind whether this is not the most acceptable theory for the majority of cases; but as already stated, I do believe that radicular lesions of the nerves occur, though in a comparatively small proportion of the total number of cases of birth injury.

When lesions in nerves occur by overstretching, it is accepted that rupture of the sheaths occurs first, permitting hemorrhage among the nerve fibers; eventually, if traction is continued, partial or complete rupture of the nerves themselves occurs. Any degree of injury, trifling or complete, may occur in this way; and it is a matter of common experience that in almost all cases of birth injury a remarkable improvement occurs without any operation, if proper non-operative treatment (massage and passive movements to prevent contractures) is begun soon after birth (Case I). Hence it is safe to assume, in the majority of cases, that the nerve lesion, wherever and whatever it is, is not very extensive, and that in such cases no operation on the nerves is advisable. But there are cases, few in number, in which to my mind the evidence of extensive nerve lesion is undoubted (Cases IV, V, VIII, X, and XIII); Sharpe speaks as if he had had experience with this class of patients alone, and at least it is to be hoped that he is not subjecting all the milder cases to exploratory operation on the nerves. He says he has operated on 56 patients in all. Unfortunately none of his cases are reported in detail, and it is impossible to judge how much improvement has been secured, and especially whether any greater improvement has been secured than may be obtained by non-operative means. Most of his operations probably were too recent for end-results to be available; but until he can show better end-results than are being secured by others without operation on the plexus, he cannot expect his views to have great influence. His chief argument in favor of early operation appears to be that even if it proves useless it will be harmless. He acknowledges that it is usually impossible to tell without direct inspection of the nerves whether or not they are seriously damaged; but he contends that it is more to the patient's interest to have an exploratory inspection of the nerves than to wait until it may be determined clinically whether or not serious nerve damage is present. In cases of apparent "total paralysis" he urges operation at the age of one month; half of those patients so treated, he says, have shown marked improvement. But it is equally true that at least half, if not more, of such patients treated without early operation also show marked improvement; it is true they do not recover nearly as good function as do those patients in whom soon after birth the apparent paralysis was not total. Those infants he has operated on at the age of three months have shown more constant improvement, because, he claims, the paralysis was less severe at first; but he believes a still greater improvement would have occurred if operation had been done at the age of one month; and he seems to urge this early resort to operation in *all* cases in the future. Older patients, he says, give the worst results. In opposition to operative measures to lessen the mechanical disability of the patient (by reducing the dislocation, if present, by tenotomies of shortened muscles, etc.) he states: "I have yet to see cured by this method of treatment one case of brachial plexus paralysis in which at birth there was total paralysis of the arm, hand and fingers." Improvement, he admits, may occur even without any treatment, but he thinks it rarely continues beyond one year of age, and a useful arm is never obtained. If he really limits his remarks to cases of "total paralysis," probably all will agree with him, but will point out that no such cases have been "cured" by neurological surgery.

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[1] These include the suprascapular (most often injured), musculocutaneous, and the circumflex; the musculospiral also is frequently injured.

[2] These include the subscapular nerves, the median and ulnar nerves, the anterior thoracic nerves (to the pectorals), and the nerves to the rhomboids and to the serratus magnus.

As regards *treatment*, then,.I reject nerve operations in early infancy. Babies are seldom if ever seen for the shoulder injury until they have reached the age of three or four weeks. At this time they either have or have not a demonstrable dislocation.

In case *no dislocation is present*, I believe the proper treatment consists, as pointed out by G. G. Davis, in keeping the hand and arm in front of the body (so as to prevent extension of the elbow and excessive internal rotation of the shoulder) by putting the arm in a sling or by pinning the sleeve to the front of the dress. In addition, as soon as the soreness of the birth injury permits, passive motions should be instituted, especially external rotation and abduction of the shoulder, to prevent shortening of the subscapularis and anterior capsule. Massage also is of value, and above all the child should be encouraged to make active movements of the kind indicated above. Electricity certainly has some value, at least in children of the age of six months or older. I think its value in early infancy is open to question. If these patients are treated in this way from the time of birth, most will recover with little permanent disability. In a few such patients, and in most of those seen for the first time a number of months after birth (especially if dislocation of the shoulder is present), additional measures will be necessary as detailed below.

In case *dislocation is present:* In some patients there can be no doubt that the dislocation was present at birth; but it is a question whether there are any examples of dislocations of the shoulder which were present *in utero*, *i. e.*, truly "congenital" dislocations.[1]

It is possible in some babies seen very soon after birth that proper treatment as above indicated will secure reduction of the dislocation by passive motions (Case IV). But in all cases where the dislocation is at all pronounced, I think it is advisable to anesthetize the child at about the age of six months, and to reduce the dislocation bloodlessly in a manner analogous to that employed by G. G. Davis in treating congenital dislocations of the hip: the baby is placed prone on a well-padded orthopedic table, with the affected extremity hanging over the side of the table. Owing to the internal rotation of the humerus the forearm will not hang vertically downward, but will point toward the patient's feet. First, this internal rotation should be overcome by very gently but persistently rotating the humerus outward by means of the flexed forearm. When the forearm has been brought thus into the horizontal plane (the coronal plane of the patient's body) direct downward pressure should be made on the head of the humerus, which usually will be felt to jump forward into normal relation with the glenoid process. Reproducing the dislocation and again reducing the head several times will leave no doubt in the mind of the operator or assistants that proper reduction has been secured. When the head of the humerus has thus been forced out onto the glenoid eminence, it will be found that the elbow can no longer be fully extended, owing to the shortening of the flexor muscles (long and short heads of the biceps and the coracobrachialis). This is analogous to the shortening of the hamstrings produced by reduction of a congenital dislocation of the hip. As in the case of the hip, so also at the shoulder, the head of the dislocated bone may be palpated anteriorly so soon as reduction is secured. It still remains to secure abduction of the shoulder-joint. This is most safely done by moving the child away from the edge of the table, so that the arm no longer hangs over the side of the table but lies upon it. With the flexed elbow lying upon the surface of the table, and the child's chest flat on the table, it will be observed that the shoulder is kept some distance above the table, by the tension of the anterior capsule and subscapularis muscles. Intermittent downward pressure is then made upon the posterior surface of the shoulder until its anterior surface comes into contact with the surface of the table. Then the elbow (still flexed) is very gradually raised from the table, by placing beneath it first one, then two, and later three or four folded towels, while intermittent downward pressure is made on the posterior surface of the shoulder. This process should be continued until the elbow lies well posterior to the frontal plane of the patient's body. There will now be little tendency for the dislocation to recur, and the entire upper extremity and chest are to be encased in plaster of Paris to maintain this position. This gypsum case should be retained for six weeks, when it should be renewed, with the arm in the same position; the second case should not be removed for six weeks more, thus maintaining reduction in the overcorrected position for a period of three months. If a shorter period of overcorrection is permitted, the dislocation tends to recur, especially in older patients. I have found that after the age of four years bloodless reduction is not efficient. It does more damage to the structures of the joint, and accomplishes less than bloody reduction by arthrotomy.

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[1] I do not know how to explain the conditions encountered in Case IV. It is difficult to explain the persistency with which the arm rebounded to the highly abnormal position in which it was found at birth unless we admit that this position had been long maintained *in utero*. Though the labor in this case was difficult ("version was attempted"), it might be that the difficult labor was caused by the malposition of the arm *in utero*, not that the labor produced this malposition. Certainly in all ordinary recent traumatic dislocations, when the dislocation is reduced the deformity does not recur immediately, as it did in this patient during a period of from three to four months after birth. However, in this patient there is no reasonable doubt that there was injury in birth, since even as late as May, 1917, when the child was in her fifth year, considerable paralysis remained, the hand being almost useless, though the shoulder and elbow were normal.

In patients over four years of age I recommend the following method for reduction of the dislocation:

A curved incision is made, as advised by Senn, around the acromion and about 3 to 4 cm. distant from it, from the coracoid process in front to the spine of the scapula behind (Fig. 71). This incision is deepened until the deltoid is exposed, and the skin and fascia are then turned upward as a flap, thoroughly exposing the acromion and the acromioclavicular joint. An incision is then made through the fascia and periosteum covering the spine of the scapula, and this spine is bared by stripping the periosteum from it until a guide can be passed under the base of the acromion, to protect the suprascapular nerve and vessels (Fig. 72). The acromion is then osteotomized obliquely at its base, the acromioclavicular joint is opened, and the acromion is turned forward, carrying with it the deltoid (modification of Kocher's method for excision of the shoulder). This thoroughly exposes the upper aspect of the shoulder-joint (Fig. 73). The long tendon of the biceps is identified; on its median or anterior border lies the lesser tuberosity, while laterally or posteriorly lies the greater tuberosity.[1] To secure normal external rotation of the shoulder, the insertion of the subscapularis is now divided while kept under tension by an assistant endeavoring to rotate the humerus outward. At once reduction of the posterior dislocation becomes possible without difficulty, and usually abduction also now becomes free; but if it is still resisted it may be necessary to divide or lengthen the tendon of the pectoralis major through a separate incision. Next, while

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[1] In several cases I have found the bicipital groove located directly in the line of the external condyle, owing to excessive internal rotation of the shaft of the humerus below the tuberosities, presumably due to the unopposed action of the unparalyzed pectoralis major and teres major muscles. Hoffa, it should be recalled, treated some of his patients by osteotomy of the shaft of the humerus about its center, rotating the lower fragment outward. This, however, does not overcome the chief cause of disability, namely, the internal rotation at the shoulder-joint, where the subscapularis is tense.

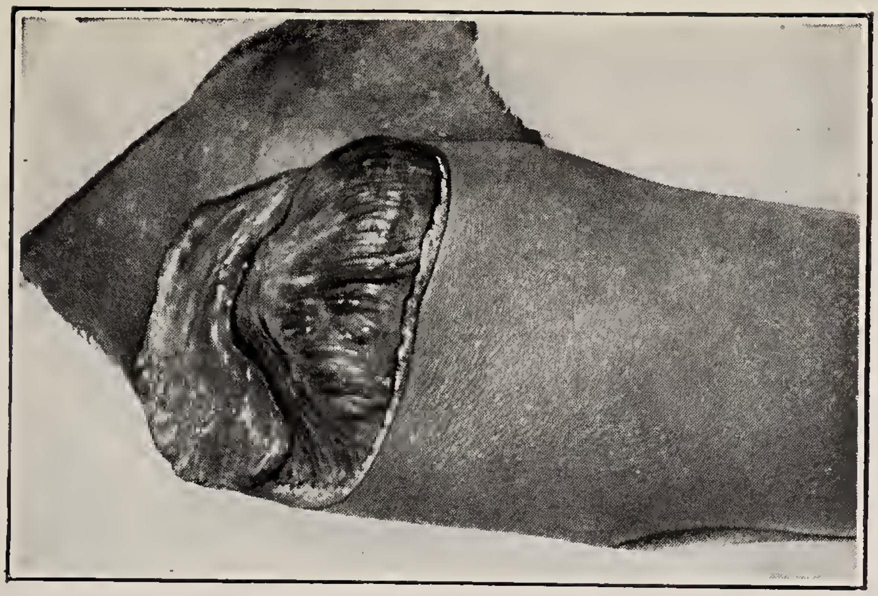

FIG. 71.—Curved incision around acromion, flap of skin turned up.

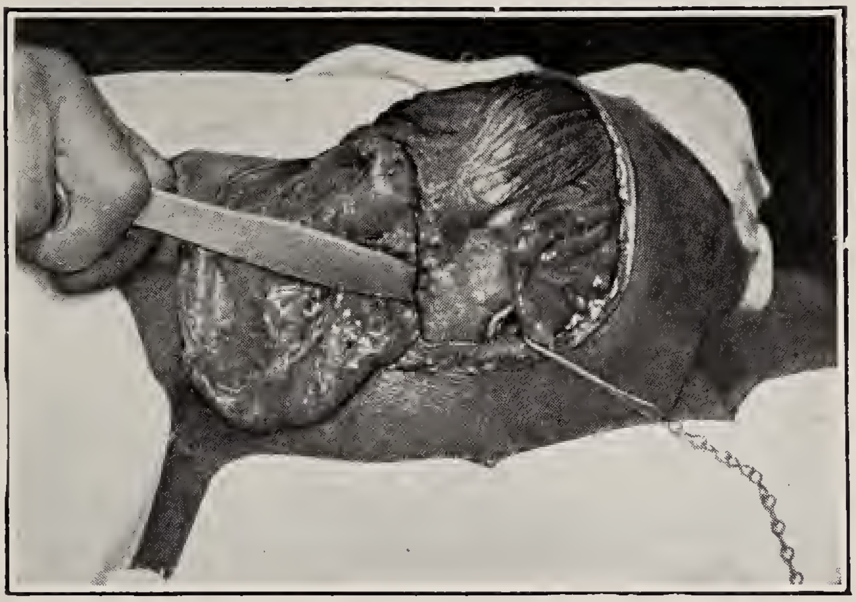

FIG. 72.—Spine of scapula exposed and retractor passed under base of acromion.

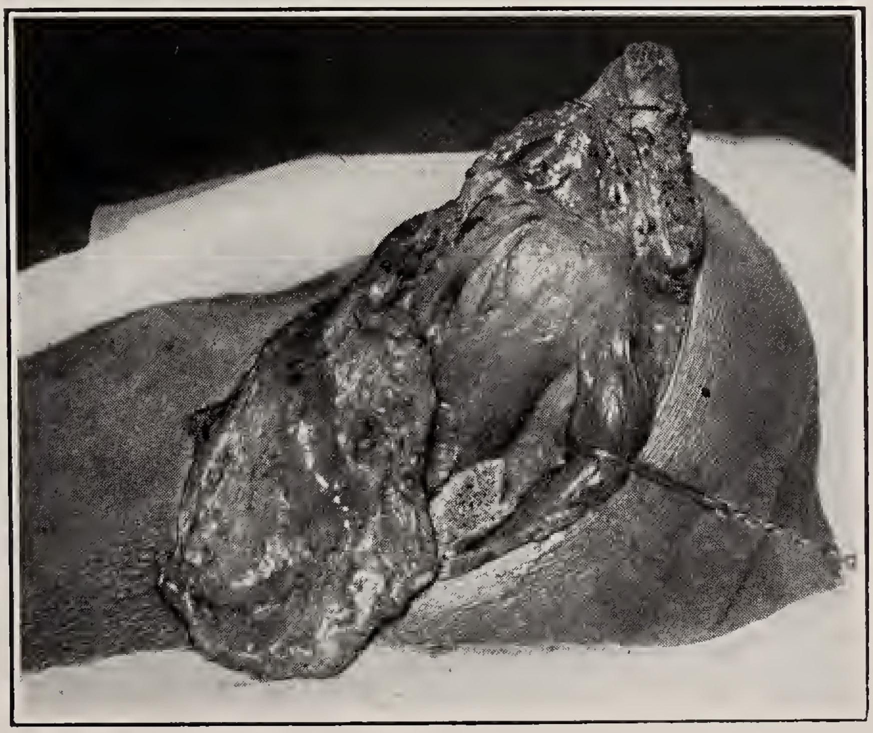

FIG. 73.—Acromion divided at its base and turned forward. The long tendon of the biceps is clearly exposed; in front of it the subscapularis just comes to view; posteriorly the supraspinatus and infraspinatus muscles are seen.

the humerus is maintained in full external rotation and abduction, the tendons of the supraspinatus and infraspinatus and teres minor muscles, as they insert into the greater tuberosity, are plicated by mattress sutures of chromic gut, thus shortening them, and maintaining the head of the humerus in its forward and externally rotated position. The acromion is now replaced. In some cases it is so much deformed that it will no longer fit in its former position, as the place its tip formerly occupied is now held by the reduced head of the humerus. It is then necessary to remove a section from the sawn surface of the acromion, in order to raise its tip out of the way of the shoulder-joint (Case X). The acromion in younger patients is largely cartilaginous and may be held in place by sutures of chromic gut including the overlying periosteum, fascia and muscles, as well as the bone; but in older patients screw fixation is preferable. The skin flap is then replaced, and sutured without drainage, the arm being constantly maintained in the "light-house position." The plaster-of-Paris dressing is not to be changed for six weeks, when a new similar fixed dressing is applied for a second period of six weeks.

After removal of the dressings it is important to resume active and passive movements, especially to encourage external rotation, abduction and supination. I have adopted this operation in five cases, with no recurrence in any patient, and marked improvement in all (Cases V, VI, VII, VIII and X).

Even in older patients where no dislocation is present, very great disability may be caused merely by muscular contractures, and this may be relieved much more rapidly and efficiently by operative means than by prolonged courses of passive movements and gymnastics. The greatest disability in all these cases usually is due to *loss of supination* and to *loss of external rotation at the shoulder.* Even when all the muscles are strong and active, the children cannot get their hands to their mouths without abducting the humerus (owing to the fixed internal rotation), and in many cases they cannot get their hands to their mouths at all, even in the position of pronation. Treatment by a series of plaster cases, endeavoring to overcome the contractures gradually, as in some contractures of tuberculous joints, has also been tried in my clinics, but has not been found so efficient as the cutting operation. For this purpose an anterior incision is made, as for excision of the shoulder, passing through the anterior fibers of the deltoid; the long tendon of the biceps is identified, and then the lesser tuberosity, where section is made of the tendon of the subscapularis. If contracture of the pectoralis major prevents full abduction, its tendon may be lengthened by Z-plasty or may be completely divided, through a prolongation downward of the same incision. I have not observed any disability to follow complete section of the tendon of this muscle, and believe it is simpler and therefore preferable to a formal lengthening operation.[1] I have employed this operation for birth injury of the shoulder in two cases (Cases XI and XII), and my assistant, Dr. A. Bruce Gill, has also adopted it with satisfaction.

In one case in the present series the disability at the shoulder arose chiefly from the paralysis of the deltoid; this was entirely relieved by transplantation of the pectoralis major to supplant the deltoid (Case XIII). In this same patient there was great disability as well from the residual paralyses in the forearm; function was materially improved by tendon transplantations.

In a fair proportion of patients with birth injury at the shoulder, who have not received adequate treatment soon after birth, considerable disability persists from deformity at the elbow. It is possible that the elbow is injured at the time of birth, but I am inclined to believe that the main deformity is gradually developed as the result of the malposition in which the elbow is held. The forearm is kept in full pronation, the elbow slightly flexed, and the whole limb internally rotated. The head of the radius gradually grows upward past the external condyle, and when seen in adolescence the patient may present a complete posterior dislocation of the head of the radius, as in Case X of the present series. All grades of subluxation may be present. It is known that in cases of hereditary deforming chondrodysplasia posterior dislocation of the radius is not unusual, owing to the lack of equal growth of the ulna; and it may be that disuse of the forearm and use of the hand (which is the usual state of function in patients with birth injury of the shoulder) tend to produce lack of development of the ulna (which is an elbow and forearm bone) and full development or even overdevelopment of the radius (which belongs rather to the wrist and hand than to the elbow or forearm).

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[1] Fairbank, in 1913, described an operation similar to this, and Sever has lately described a modification of it as an original procedure; his modifications consist in making the incision in the deltopectoral groove (where the cephalic vein is in the way), and in dividing the tendon of the subscapularis on a grooved director.

The case histories which follow have been selected to illustrate the prognosis and treatment of certain types of the affection:

1. The usual mild case, recovering nearly perfect function under treatment by massage, passive and active movements, etc. (Case I).
2. The same type of case, but complicated by posterior dislocation of the humerus (Cases II and III).
3. The severe type, in which the residual paralysis causes far more disability than could any dislocation (Cases IV and V).
4. The typical case of subspinous dislocation coming under treatment late, and with no disability except that due to the joint deformity (Case VI).
5. The typical case of subspinous dislocation with moderate paralytic disability (Cases VII, VIII, IX, and X).
6. The typical case of disability from contractures without either dislocation or paralysis (Cases XI and XII).
7. The unusual type of flail shoulder (Case XIII).

The frequency of subspinous dislocation in the patients under my care is worth noting; it was present in more than 40 per cent.

Another feature which I think bears emphasis is the frequency with which two or more children in the same family are affected: in one instance three sisters were similarly injured in birth, and the fourth child was killed during delivery. In several other instances brothers or sisters have died at or soon after birth from injury; in one case (Case VI) 5 other children were killed at birth. There is room here for improvement in obstetrics.

CASE I.—*Birth injury of the right shoulder without dislocation; recovery under treatment by massage and active and passive movements.* Mollie R. was brought to the Orthopædic Hospital October 10, 1914, at the age of ten weeks. It had been a footling presentation, easy labor of four hours' duration, no instruments being used. There was one older child, now three years old.

Examination. The right shoulder is held quietly by the side, in internal rotation. She makes no attempt to move the shoulder, but she moves the fingers slightly. No dislocation is present. The electrical report by Dr. Cadwalader shows that the deltoid does not react to Faradism, and Galvanic current causes too much pain to be used.

Treatment. Massage and passive motions, three times weekly.

November 26. Has fair grasp in hand, uses arm a little. It can be passively raised above head and placed behind back. External rotation is limited a little within the sagittal plane.

January 9, 1915. External rotation is possible beyond the sagittal plane.

February 13. Nearly seven months old. Dr. Cadwalader reports: probably complete reaction of degeneration in the deltoid; no reactions of degeneration in other muscles.

June, 1915. Uses hand and arm all the time.

August, 1915. No dislocation has developed. Fair power has returned in the deltoid.

December, 1915. Puts hand to mouth in semisupination, and with shoulder slightly abducted. Scarcely any limitation of passive external rotation or abduction.

July, 1916. Can raise right arm above head almost as well as left arm. Puts hand to mouth and top of head easily, and can be placed behind back passively with ease.

October, 1916. Right arm almost normal. Dismissed from further treatment at the age of twenty-six months.

CASE II.—*Subspinous dislocation of right humerus; bloodless reduction at age of six months.* William A. F. was first seen at the Orthopædic Hospital November 6, 1913, at the age of three months. He had one sister, six years old, who was normal. This boy had been injured in birth which was not instrumental (L. O. A.). There had been a cephalhematoma at birth, over the right parietal, and this region was still palpably thickened up to the age of four months. He held his right arm at the side, in internal rotation, and all shoulder motions were limited, especially abduction. (Fig. 75 *A*). He has pain when the arm is moved. The hand, fingers and wrist are kept flexed, and the hand is in ulnar adduction. There is good power in the triceps, and a good grip in the hand, but no power of flexion of the elbow. The head of the humerus was palpable beneath the spine of the scapula, and a skiagraph (Fig. 74) showed the epiphysial center for the head of the humerus not opposite the glenoid as on the normal side, but luxated out and up. As the child was brought from out of the city, the mother was directed to make the necessary manipulations at home.

December 4. The shoulder still is tender. When the humerus is abducted and externally rotated to the limit and pressure is made forward on the head of the humerus a distinct grating click or snap can be felt as the head slips over the posterior border of the glenoid. Active flexion of the elbow has developed

Febuary 5, 1914. Head of humerus still dislocated posteriorly, so on February 7, under ether anesthesia, bloodless reduction was done, and the arm dressed in the light-house position. The child was taken home the same day, and the mother later reported that almost at once he began to show better use of his hand. Three weeks later it was noted that fair power of extension of the fingers had developed.

March 28. New gypsum case was applied, the humerus remaining in proper position.

May 7. Gypsum dressing permanently removed, the shoulder remaining reduced.

May 28. Since last note mother says he has been using the hand "just like normal." External rotation still limited at sagittal plane.

August 8. Can get hand to mouth, but only in pronation.

January 23, 1915. Uses right hand more than left, and beginning to hold his hand in supination. Active and passive exercises have been continued all the time.

March 20. Good extension in fingers, but none yet in wrist.

January, 1916. As progress seemed slow during the past year, the arm was again dressed in full abduction and external rotation in plaster of Paris (applied without any anesthetic, at the limits of movement), and this dressing was worn for seven weeks. The power of supination and external rotation was decidedly improved when the cast was finally removed.

July, 1916. It was noted that the power of extension of the fingers and wrist was good.

December 30, 1916. Still prefers to use the left hand. Active movements: supination not quite complete, external rotation of shoulder only to sagittal plane; abduction and elevation of upper extremity about 30 degrees short of vertical. Passive movements: supination complete, external rotation about 20 degrees beyond sagittal plane; abduction (without elevation of scapula) about 50 degrees (limited by axillary fold muscles). There was a slight hollow beneath the acromion in front, and the head of the humerus was just palpable posteriorly beneath the spine of the scapula (subluxation).

March 10, 1917. The accompanying photograph (Fig. 75 *B*) was made, showing the boy putting his hand to his mouth.

CASE III.—*Birth injury of right shoulder, first seen at age of seven weeks. Dislocation of shoulder not observed until age of three years.* Ida May F. came to the Orthopædic Hospital when seven weeks of age, June 8, 1911. This was the first baby in the family, a head presentation, instrumental delivery.

Examination showed the right arm hanging by the side in internal rotation. The hand grasp was good; the anteroposterior motions (flexion and extension) of the shoulder were good, and abduction was fair. She could not flex the elbow. Passive motion was normal throughout, and there was no pain on motion. The epiphysis rotated with the shaft. There was some crackling in the region of the shoulder-joint.

Treatment consisted in bandaging the arm in the Velpeau position for two weeks; then massage, passive movements and electricity were given.

October 5, 1911. Can flex elbow, and has more use of shoulder, but is still in internal rotation.

March 28, 1912. Slight improvement in strength.

June 11, 1914. Careful reëxamination reveals a posterior subluxation of the humerus at shoulder, with a hollow beneath the acromion. The use of hand and elbow are good. There is still limited abduction and external rotation at the shoulder. (It is highly probable that a more attentive examination soon after birth would have detected the posterior luxation of the shoulder; atten-

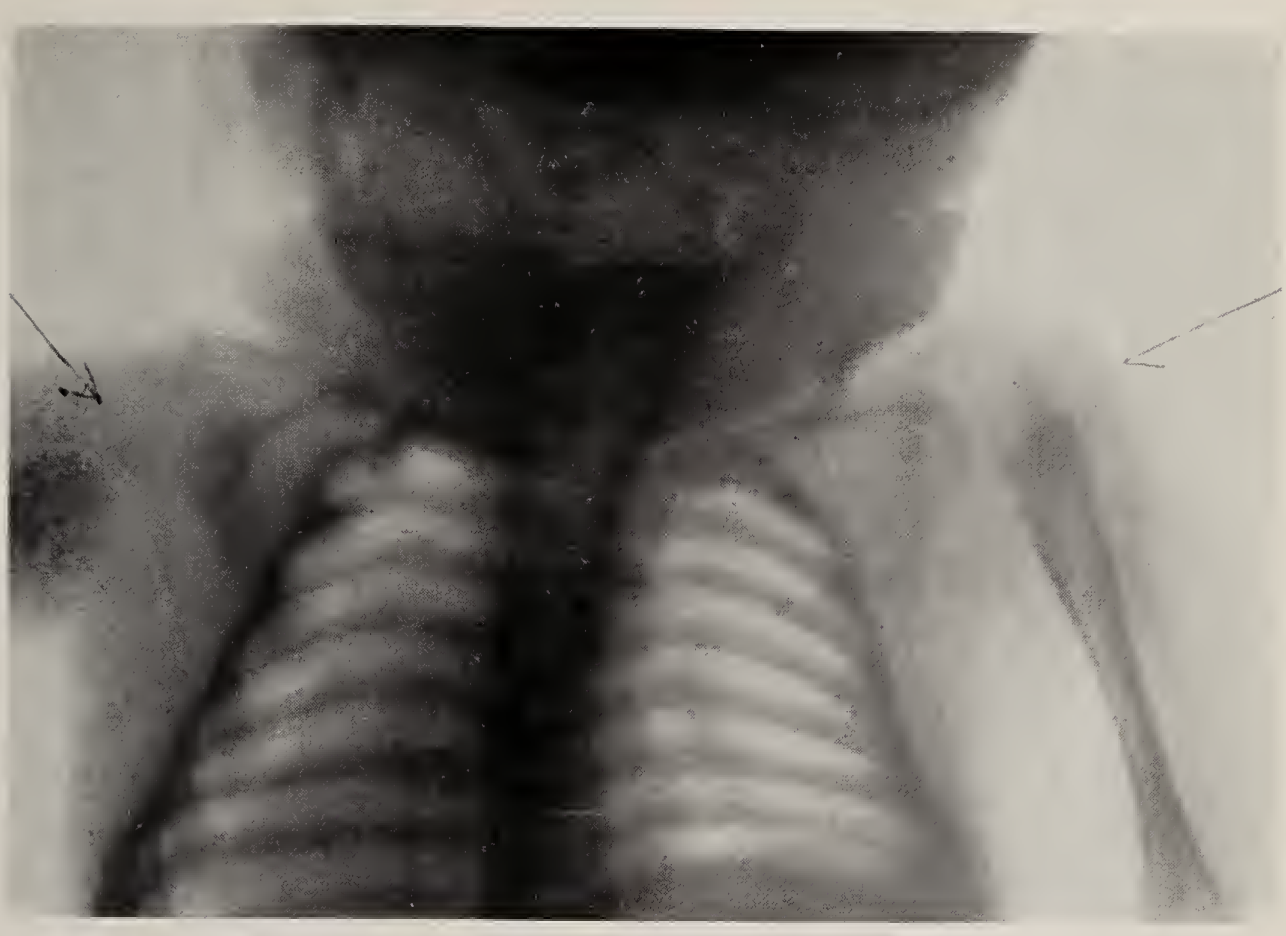
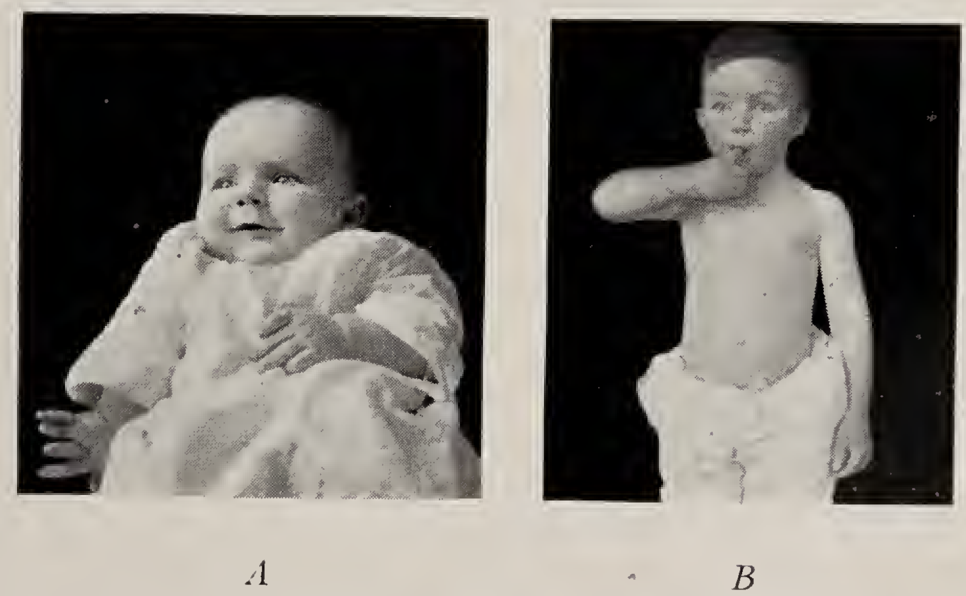


Fig. 74.—Case II. Subspinous dislocation of right humerus; patient aged three months. Note that on the injured side (arm held close to body in internal rotation) the epiphysis of the head of the humerus is far removed from the glenoid.



A *B*

Fig. 75.—Case II. *A*, aged six months; *B*, aged three and one-half years.

tion was now directed more particularly to this deformity on account of T. T. Thomas's paper.)

March 13, 1917. The child is now six years old. She was treated by massage and passive motions until two years ago. She now gets her hand to her mouth only with the forearm in mid-pronation and with the arm abducted. She gets her hand to her back easily and fastens her own petticoat. Supination is weak. There is no external rotation at the shoulder beyond the sagittal plane. A slight posterior subluxation of the shoulder persists. She uses her right hand normally, but prefers to use the left. Function could be improved by tenotomy of the subscapularis, and perhaps of the pectoralis major.

Case IV.—*Subspinous dislocation (luxatio erecta) of left humerus from birth injury; persistent paralysis of hand.* Agnes T., ten weeks old, seen at Orthopædic Hospital, March 7, 1913. This is the fourth child, the previous births being normal. In the present instance "version was attempted" and at birth the left forearm was folded across the front of the baby's neck, in full pronation, and it has constantly rebounded to this position ever since when not held down by a bandage. The mother says that when two weeks old a long splint from axilla to palm was applied by the family physician, and was kept in place for two weeks; and that when it was removed the child could not move her fingers as well as when first born.

Examination at the age of ten weeks: the arm is held abducted to 90 degrees, and the head of the humerus is palpable beneath the spine of the scapula; there is some grating on attempts at rotation. A bony lump in the axilla is thought to be the glenoid. There is no active power to extend the fingers, wrist or elbow.

Two or three weeks later, the arm having been bandaged to the side constantly, it was noted that the arm stays almost against the side without being held down, and it is assuming the typical posture of "obstetrical palsy."

August, 1913. Now eight months old: the arm comes easily down to the side, and the head of the humerus stays in the glenoid. Wrist drop and paralysis of the triceps persist. Fair power in biceps, and fair grip in fingers; no power in axillary fold muscles; deltoid is doubtful.

October, 1913. Head of humerus is clearly anterior to acromion and there is no palpable deformity as compared with the uninjured side. Grip is good, the hand as a rule staying clenched and the wrist flexed, but not so persistently as before. No power yet in musculospiral; some power in pectoralis major. Dr. H. P. Boyer reported that the electrical examination of the muscles was a little doubtful, but he thought there was no reaction of degeneration in the pectorals or the extensors of the fingers. There was "some response to the Faradic current."

June, 1914. Very little further improvement has occurred. Massage and electricity have been given twice weekly. She can now abduct her shoulder (deltoid) to 90 degrees; the axillary fold muscles are good, and the biceps is good. There is no power in the triceps or in the extensors of the fingers; there is very slight power in the flexors of the fingers. The fingers are still held clenched and the wrist flexed. Passive movements: external rotation at the shoulder is easy to beyond the sagittal plane; the elbow can be extended to 170 degrees, supination is normal, but pronation a little limited. The mother says the child has been *biting* her hand (not very hard) for four or five months. A posterior splint was applied to keep the fingers and wrist in extension, and all other treatment was suspended.

August, 1914. Does not bite hand any more; hand no longer stays flexed, but there is no power in the fingers nor in the extensors of the wrist. The posterior splint was continued.

November, 1914. Has learned to hold a glass of milk or water in the bent elbow and drinks easily from it.

January, 1915. Burned the left elbow a few days ago, and it seemed to give her no pain. The finger nails of the paralyzed hand require to be be cut about three times as often as those of the normal hand, the latter being worn down by use.

March, 1917. Now four years of age. The hand is useless—in flexion and ulnar deviation; she can barely flex the fingers. The biceps and triceps have very fair power. She gets the back of her wrist to her mouth, and holds articles in the bend of her elbow with great security.

CASE V—*Subspinous dislocation of humerus, with complete flaccid paralysis of arm; reduction by arthrotomy.* Catharine B. came to the Episcopal Hospital in September, 1913, when past three years of age (Fig. 76). There were two older children: the first (now seven years old) had the left shoulder "broken in birth"; the second child (now six years old) had "the nerve hurt" in birth (right shoulder), but "both came all right about six weeks after birth." (See Cases V*a* and V*b*). A fourth child, born subsequent to this visit (in May, 1914) was killed during delivery.

Catharine was born head first, and no instruments were used. She seemed to have no sensation in the left arm. It has always been utterly useless. She chews the fingers, and has burned the hand several times without evincing any sensations of pain.

Examination. There is complete flaccid paralysis of the left upper extremity; probably, it was thought, the lesion was a tearing out of the roots of the brachial plexus from the spinal cord. She has been seen by nerve specialists and orthopedic surgeons, who told the mother nothing could be done. Owing to the complete absence of sensation, persisting for over three years, this seemed reasonable advice, and it was repeated.

June 15, 1914: Nine months after the first examination the child was sent for, for reëxamination, especially as it had not been noted at the first examination whether or not there was a posterior dislocation of the shoulder. Today it is learned that the child has not burned her fingers since Christmas, and that soon after the first of the year she began to move her fingers. She can now flex the shoulder slightly. The axillary muscles have fair power; there is some power in the biceps, and she can extend the wrist well. There is a subspinous dislocation of the humerus. Operation was now recommended to secure reduction of the dislocation, since it had been determined by an experience with six cases of reduction of similar dislocations (three "bloody" and three "bloodless" reductions), all treated since this patient was first seen, that a considerable degree of improvement might be expected in apparently paralyzed muscles if deformities were overcome and the weak muscles were allowed to work at a better advantage.

Operation (December 28, 1914) Patient now four years of age. Senn's incision, with temporary resection of the acromion. The acromion was cut at its origin from the spine by an osteotome, but in turning it forward the acromion fractured through its epiphysial cartilage. The intervening detached piece of acromion was laid aside in dry sterile gauze and was re-implanted at the conclusion of the operation. The tendon of the subscapularis was divided, thus allowing external rotation of the humerus until the flexed forearm was in the coronal plane. The head of the humerus was thus easily and fully reduced to its normal position. The tendons of the supra- and infraspinatus muscles were then shortened; the detached piece of the acromion was replaced and fastened to the spine of the scapula by a small Lambotte screw; the cartilaginous tip of the acromion was turned up again and sutured to the periosteum covering the head of the screw; and the fascia and skin were closed with interrupted chromic gut sutures. The arm was dressed in abduction and external rotation, in plaster of Paris.

January 9, 1915. Went home.

February 15, 1915. New case applied. One granulation in the line of incision.

March 29. Case removed. Massage ordered. Incision healed.

April 10. Can get hand to mouth, but only with arm in abduction. Has fair power of flexion and extension in elbow, but scarcely any motion in hand.

November 15, 1915. Dislocation stays reduced; the acromion is solid. She can put her hand to her mouth with forearm in pronation and the humerus abducted. She has no active external rotation; has slight power in the triceps, can flex elbow to 45 degrees, and there is passive extension of the elbow to 150 degrees. She has some power in the extensors of the wrist, very slight power in the thumb, fair pronation and supination. There is no power in the other muscles of the hand.

CASE V*a*.—*Subspinous dislocation of left shoulder; slight disability.* Anna B., aged seven years, is the eldest sister of Catharine B. (Case V). It was a head presentation at birth, and no instruments were used; but after the head was delivered, the shoulders stuck, and the labor was difficult.

Examination. The mother brought this child to the Episcopal Hospital, June 15, 1914, by request. Except for the fact that the baby's "shoulder had been broken in birth" it was thought that she had recovered perfectly by the age of six weeks. Examination showed, however, that there was a subspinous dislocation of the humerus, with the characteristic internal rotation of the arm, which was carried into marked abduction on putting the hand to the mouth. Treatment was declined by the parent, and was not urged, as the disability was slight (Fig. 76).

CASE V*b*.—*Birth injury of right shoulder, without dislocation of the humerus.* Mary B., aged six years, was the sister of the patients already described (Cases V and V*a*). She was brought to the Episcopal Hospital for examination, by request, June 15, 1914. There was the characteristic attitude of carrying the injured limb, with the arm in internal rotation and the forearm pronated. No dislocation of the shoulder was present, but there was limitation of external rotation and of supination, and in carrying the hand to the mouth the arm was forced into marked abduction. The disability was so trifling that no treatment was recommended (Fig. 76).

CASE VI.—*Subspinous dislocation of humerus, reduction by arthrotomy.* Theresa M. came to the Episcopal Hospital when five years old, April 5, 1915. She was the youngest of seven children, all but one of the six others having died at birth. In the instrumental delivery of Theresa, the right arm was injured.

Examination. The arm is held in internal rotation with the elbow flexed; the head of the humerus is absent from its normal site

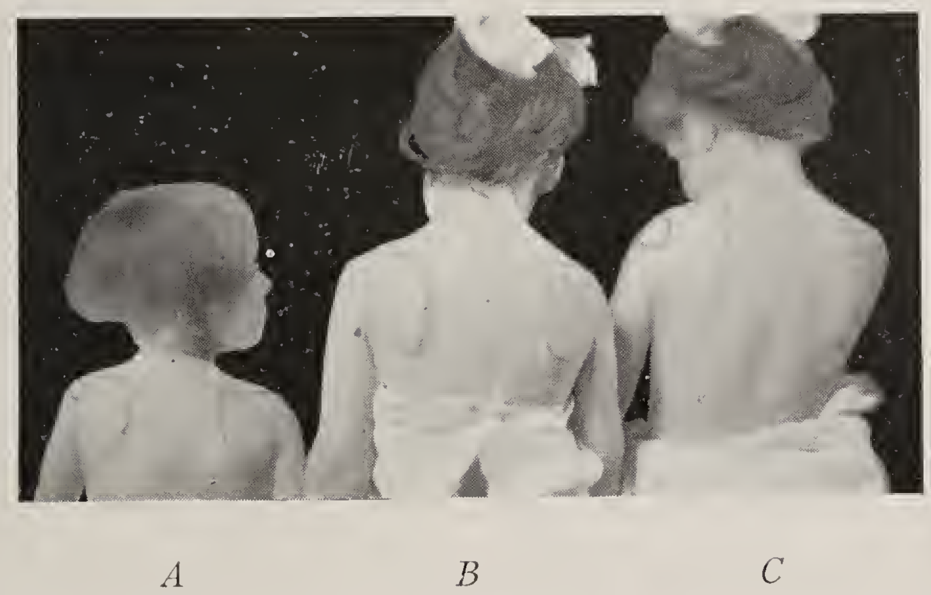


FIG. 76.—Three sisters with birth injuries of shoulders. (Fourth child killed in delivery.) *A*, child aged three years, dislocation of left shoulder; *B*, child aged six years, right shoulder (no dislocation); *C*, child aged seven years, dislocation of left shoulder.

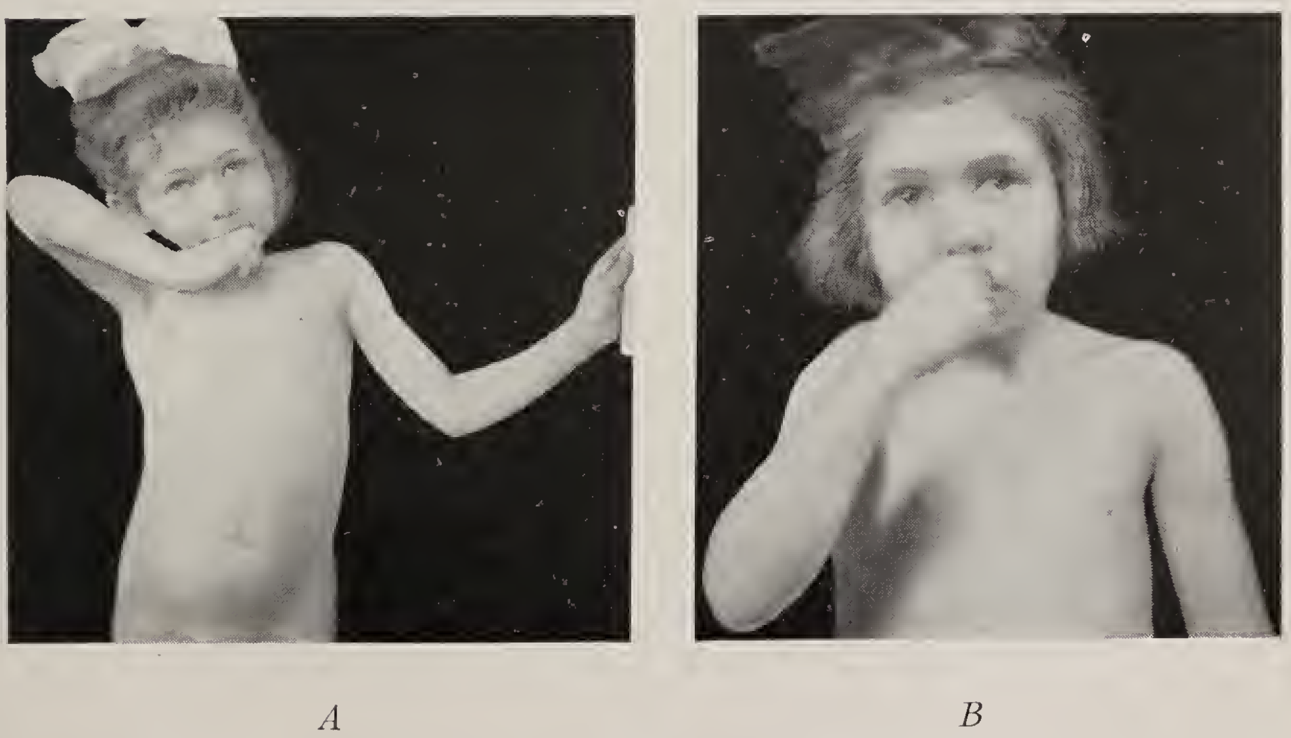


FIG. 77.—Case VI. *A*, child aged five years, posterior dislocation of right shoulder from injury at birth; *B*, eight months after operation.

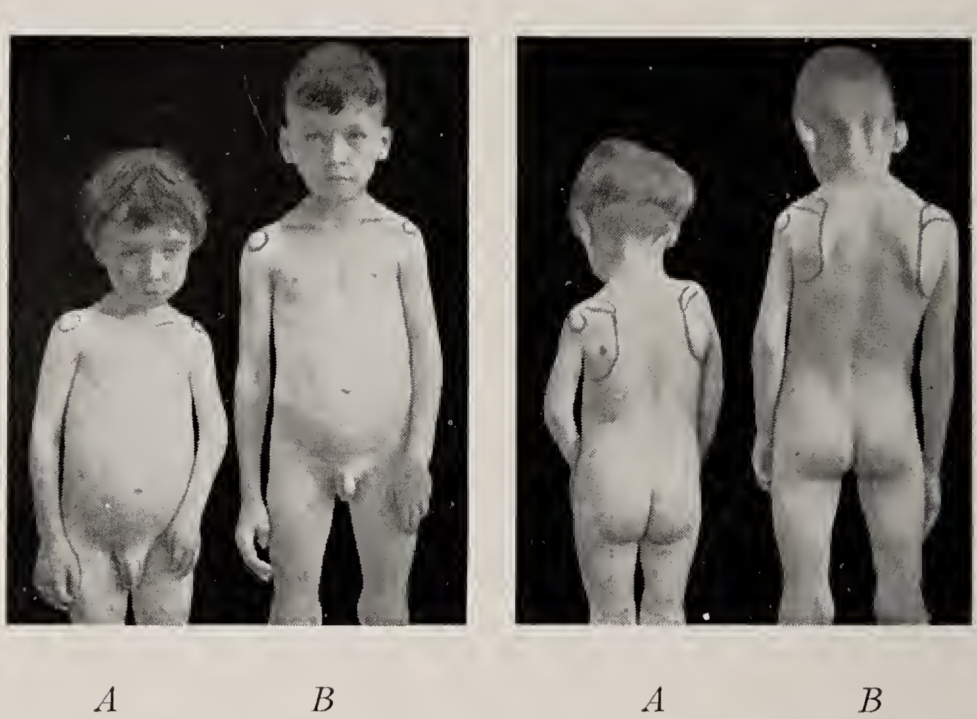


FIG. 78.—Cases VII and IX. Brothers with birth injuries of left shoulders. *A*, child aged four years; *B*, child aged seven years.

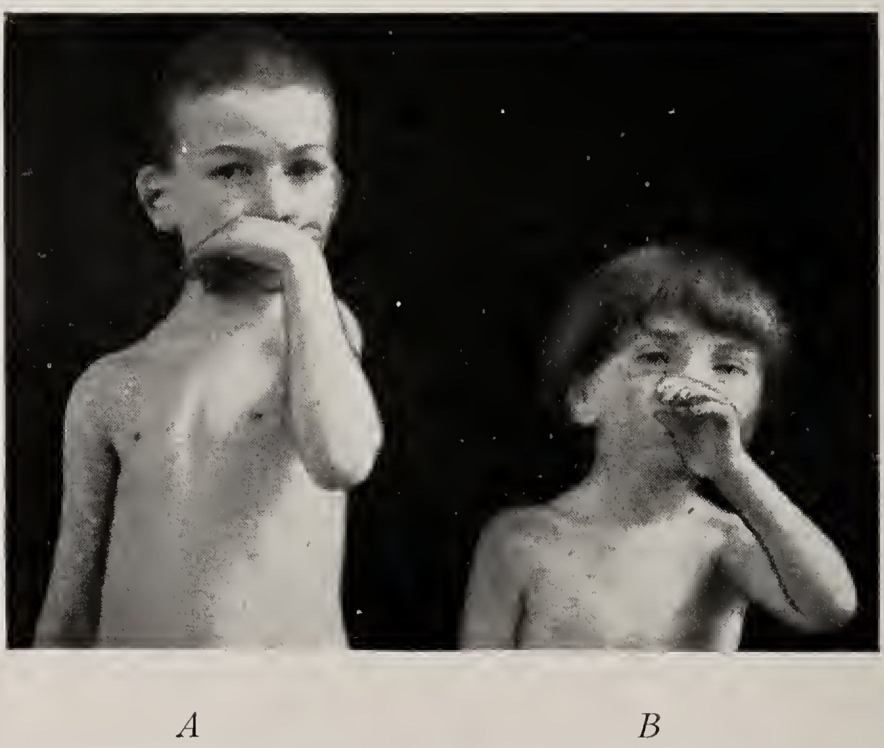


FIG. 79.—Cases VII and IX. Seven months after operation. *A*, "bloody"; *B*, "bloodless."

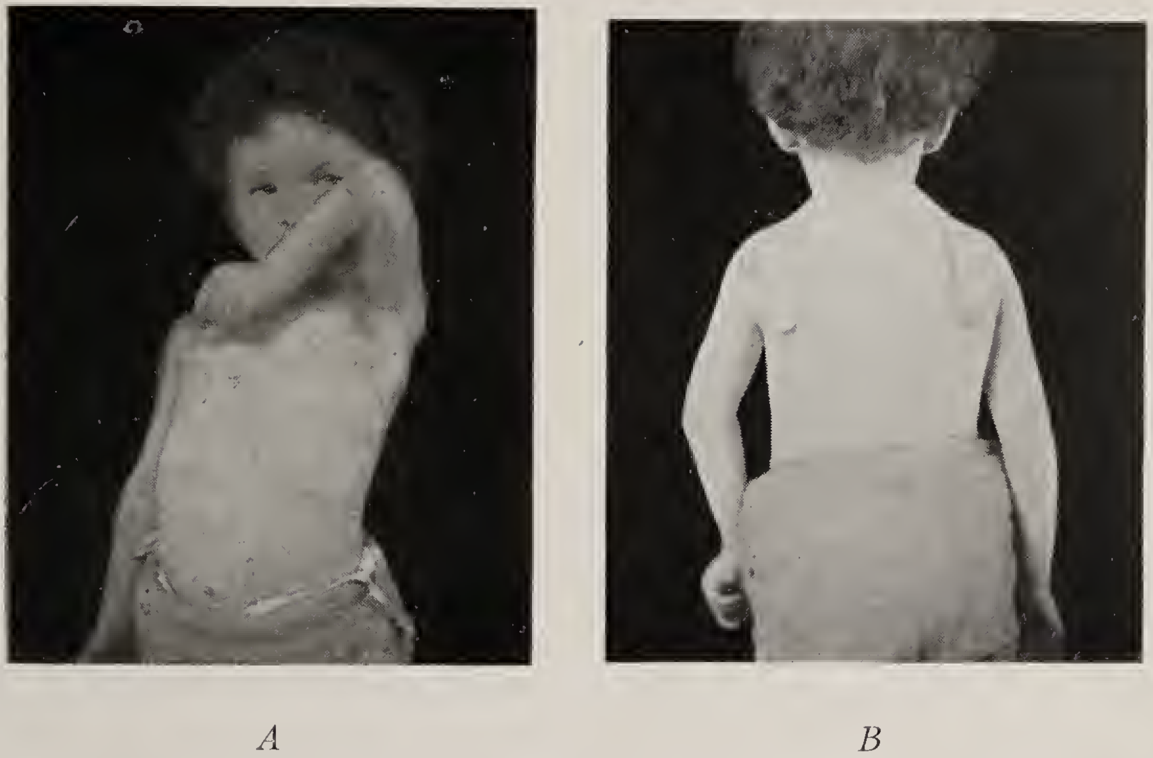


FIG. 80.—Case VIII. Child aged five years. Subspinous dislocation of left humerus. *A*, attempt to put hand to mouth; *B*, rear view.

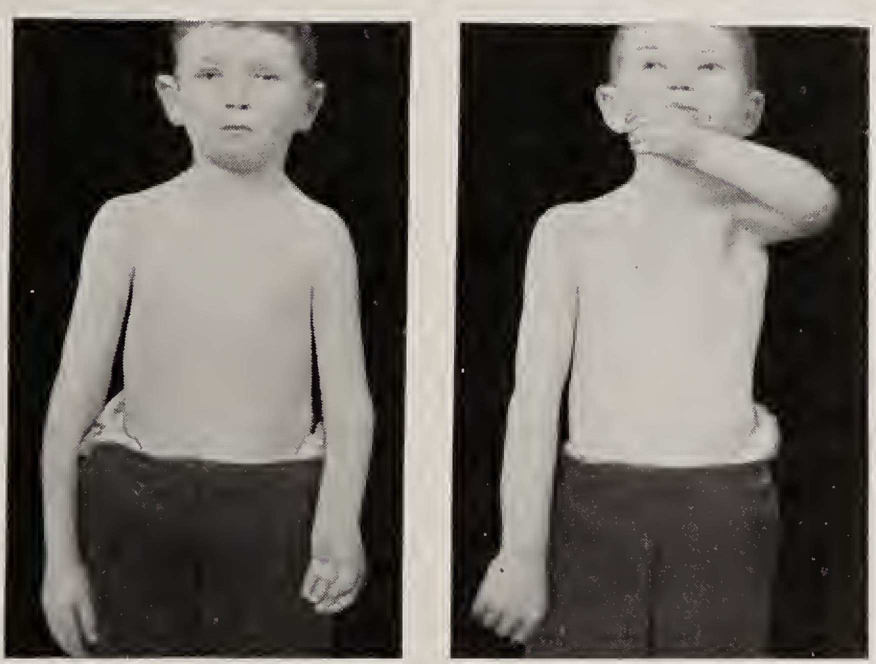


FIG. 81.—Case VIII. Child aged seven and one-half years. Birth injury of left shoulder. Two years after open operation.

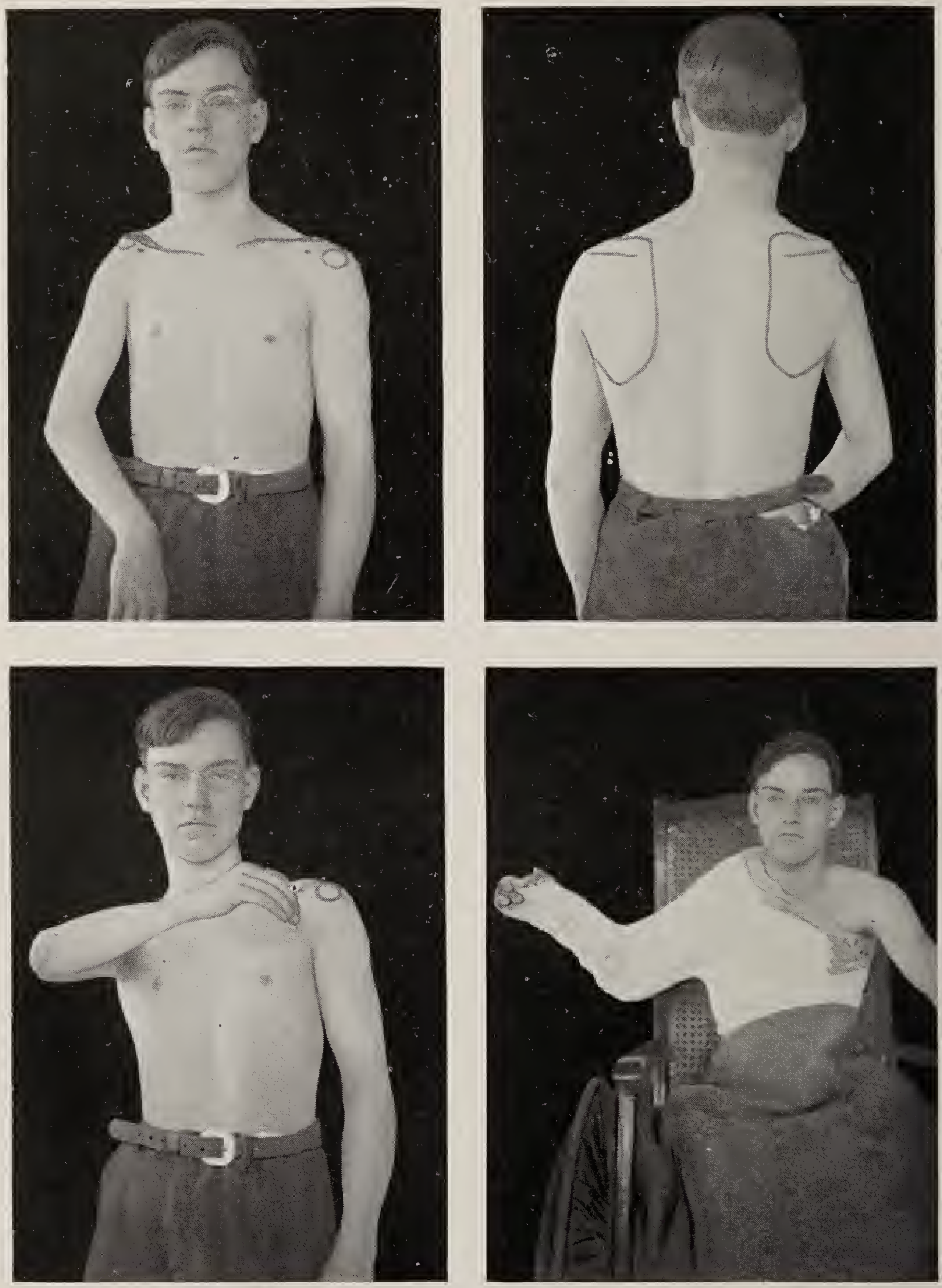


Fig. 82.—Case X. Patient aged fifteen years. Subspinous dislocation of right humerus. After reduction the arm was dressed in the most stable position.

in front of the acromion, and is palpable posteriorly beneath the spine of the scapula. She cannot put her hand to her mouth in supination, and in the act the arm is abducted until the elbow is higher than her ear (Fig. 77).

Operation (April 14, 1915). Senn incision. Acromion detached and turned down. After section of subscapularis tendon the head of the humerus was replaced in the glenoid; the tendons of the supraspinatus and infraspinatus were plicated to shorten them and maintain the external rotation; and the acromion was replaced and held in position by sutures of chromic gut. The arm was dressed in plaster of Paris in full abduction and external rotation.

April 22, 1915. Went home.

May 28, 1915. New plaster case applied.

July 12, 1915. Plaster case removed permanently. Reduction is maintained, but shoulder is stiff and painful.

August 2, 1915. Can get hand to mouth in supination, but motion in shoulder is only about half normal. Continue massage and passive motion.

December 6, 1915. Nearly perfect result (Fig. 77). Can put hand to mouth in supination, and behind head; cannot put it behind back. Extension of elbow to 150 degrees. Has good supination in forearm, has almost normal external rotation, and can abduct arm to 90 degrees.

CASE VII.—*Subspinous dislocation of shoulder; bloodless reduction.* Edward J., four years old, is a brother of Case IX. Three sisters are normal, but the third boy, born February 7, 1916, by instrumental delivery, was killed during birth (his weight is said to have been 16 pounds). When the present patient was examined, in September, 1914, there was a subspinous dislocation of the left humerus, slight cubitus varus, and limited rotation in the forearm. The arm was not so useless as that of the patient's brother (Case IX), but like Tom he could not get his arm into external rotation, nor his forearm into supination (Fig. 78).

Operation (September 8, 1914). Bloodless reduction, under ether anesthesia; with the child lying on his abdomen, and the arm in abduction and the forearm hanging over the edge of the table, pressure was made downward on the head of the humerus, at first with the arm in internal rotation; then gradually, *after* reduction had been secured (ascertained by palpating the head of the humerus in front of the acromion, and observing that the flexors of the forearm had become tense, as do the hamstrings when a congenital luxation of the hip is reduced), the humerus was worked into external rotation. When complete reduction was effected, an upward dislocation of the acromial and of the clavicle was produced. The limb was dressed in plaster of Paris, with the humerus horizontal in the coronal plane, and the forearm vertical (full abduction and external rotation of the shoulder). The reduction was attended by a distinct jump and click, was easily reproduced, and again reduced, with characteristic jump and click.

November 6, 1914. Case removed eight weeks after reduction, which was still present.

November 9. Dislocation has recurred.

November 11. Bloodless reduction under ether anesthesia, as before; dressed in plaster-of-Paris in same position.

December 28. New case applied. Shoulder stays reduced.

February 12, 1915. Case removed, three months after second reduction. Humerus remains in place.

April 10. Good use of hand, but humerus does not stay very far forward. Can get hand to mouth without much abduction of arm, but not in supination (Fig. 79).

March 13, 1917. Two years and four months after reduction. Has had no treatment for two years. He is nearly seven years old. He can get his hand to his mouth only in pronation, barely to his head, and not at all to the small of his back. Passive external rotation of the shoulder is possible until the flexed forearm lies almost in the coronal plane; active external rotation is limited at 45 degrees (midway between sagittal and coronal plane). Passive supination of the forearm is limited just beyond the midposition; active supination stops just short of the midposition. His arm and hand are very useful and still improving.

CASE VIII.—*Subspinous dislocation of humerus; reduction by arthrotomy.* Joseph B., five years old, came to the Episcopal Hospital February 2, 1914. He was the fourth child of his parents, three older and one younger children being normal. The fifth child was delivered instrumentally, and died in two hours. Patient was also a case of instrumental delivery, and his left arm has been paralyzed ever since birth.

Examination. He holds the left upper extremity in internal rotation and slightly abducted at the shoulder, flexed at the elbow, and hyperextended at the wrist (Fig. 80). When trying to put hand to mouth he raises arm in abduction and internal rotation, and the flexor surface of the forearm is carried against the mouth (Fig. 80, *C*). He cannot get his hand to his mouth, and the hand is useless. The head of the humerus is absent from its normal location in front of the acromion and is visible and palpable behind the spine of the scapula. His grip is good, but he cannot flex his wrist. The biceps is good, and there is slight power in the triceps. There is very slight power in the extensor carpi radialis. He cannot flex the index finger as well as the others, nor the thumb; and he cannot extend his fingers. Elbow cannot be extended beyond 160 degrees, and there is cubitus varus (195 degrees, *i. e.*, the forearm falls 15 degrees to the inner side of the axis of the humerus). The axillary fold muscles and the trapezius are good.

Operation (February 7, 1914). Senn's incision; temporary resection of acromion. Lesser tuberosity could be only very imperfectly exposed until after division of subscapularis tendon, which allowed external rotation of humerus, thus bringing lesser tuberosity into view. The head of the humerus could now be pushed forward in front of posterior lip of glenoid, and dislocation satisfactorily reduced. The supraspinatus and infraspinatus tendons were then shortened by plicating to maintain external rotation; the acromion was replaced and fixed in position by chromic sutures, and the skin wound closed. Dressed in external rotation and abduction (Fig. 80, *D*).

February 28. Went home.

May 20. Case removed.

June 1. Massage and passive motions ordered.

August 31, 1914. Shoulder remains in joint. Better motion of the arm. Splint to hold wrist slightly flexed.

January 17, 1916. Has full external rotation, and almost normal supination. He can just put his hand to his mouth in supination (Fig. 81). He cannot put it behind his head or behind his back.

March 13, 1917. Now eight years old, over three years since operation. Puts hand to mouth normally, but wrist remains in hyperextension. There is a strong grip in the fingers. Active flexion of the elbow is normal, but extension is impossible beyond 135 degrees (passive extension to 150 degrees). There is no active rotation in the shoulder, nor any active supination or pronation in the forearm. There is good power in the deltoid. He uses his hand constantly for holding things.

CASE IX.—*Subspinous dislocation of humerus; reduction by arthrotomy.* Tom J., aged six years, with his brother Edward, aged three years (Case VII), similarly deformed, was brought by his mother to the Episcopal Hospital January 22, 1913. After this date the patients were not seen until September, 1914. Boy's left shoulder had been injured in birth, and he has never had any use of the arm. Three sisters are normal, but the third boy, born February 7, 1916, by instrumental delivery, was stillborn (weight said to have been 16 pounds).

Examination (September, 1914). The left arm hangs in internal rotation, with the forearm in pronation, and is several inches shorter than the right arm (Fig. 78). Finger movements are normal, but there is no active power of extending the wrist, and passive extension is possible only to 165 degrees. The elbow can be extended to 160 degrees, and flexed normally; there is cubitus varus. The head of the humerus is palpable behind the spine of the scapula, and the tip of the acromion is turned down. Active abduction at the shoulder (including rotation of the scapula) is possible to 75 degrees, and passively to 150 degrees.

Operation (September 8, 1914). Senn's incision, with temporary resection of the acromion. The shoulder-joint was opened above the greater tuberosity, which was the only portion accessible. The head of the humerus lay against the posterior lip of the glenoid process, in posterior subluxation; and where the edge of the glenoid impinged on the humerus a deep longitudinal groove had been worn in the cartilaginous head of the humerus. It now became possible to reduce the luxation by forward pressure, if the humerus was in internal rotation, but impossible if in normal position of rotation, and also impossible to rotate the humerus out after reduction was secured. Therefore the subscapularis tendon was cut from within the joint. At once reduction became easy when the humerus was in external rotation. While the humerus was maintained in reduction, with the arm abducted and externally rotated, the tendon of the supraspinatus was plicated, to aid in maintaining abduction and external rotation; the acromion was replaced and held in position by sutures of chromic gut uniting the deltoid, periosteum and trapezius over it. The skin was similarly closed with chromic gut. The arm was dressed in plaster of Paris in the usual position of abduction and external rotation.

September 14. Went home.

November 6, 1914. Plaster case removed.

December 7, 1914. Humerus stays reduced. Arm no longer in internal rotation. Fair power regained in musculospiral nerve. Passive movements normal, except extension of shoulder, which is absent. He can get his hand to his mouth in full supination (Fig. 79, *A*).

April 16, 1915. Humerus remains reduced. Gets hand to mouth very well. Very little disability.

February 21, 1916. Humerus remains reduced. External rotation normal. Deltoid feeble. Cannot raise hand above nor behind head. Can put hand to mouth, but not in *full* supination. Passive abduction in shoulder 60 degrees. Some weakness still in extensors of wrist and fingers. Triceps and biceps good. Supination: active, one-half normal; passive, normal. Does not use the arm very much.

March 13, 1917. Mother reports the child died from appendicitis in January, 1917. She says the arm was "just grand."

CASE X.—*Subspinous dislocation of humerus, deformity of elbow, and wrist drop; arthrotomy of shoulder; excision of head of radius; arthrodesis of wrist by bone transplant.* George W. was fifteen years of age when he came to the Episcopal Hospital May 24, 1914. He had three brothers and two sisters living and well. His right shoulder had been injured in birth, and his arm was practically useless.

Examination. His right arm hangs in internal rotation, the humerus abducted, the elbow flexed, the forearm in pronation. The head of the humerus is absent from its normal site in front of and below the acromion and is visible and palpable below the spine of the scapula. Passive movements: these are normal in the fingers and hand, and in the wrist, except adduction at the wrist which is lost. Extension of the elbow to 145 degrees, and flexion to 50 degrees. Complete pronation and supination are both lacking, rotation being present only through an arc of 30 degrees, most limited in supination. The head of the radius is prominent beneath instead of anterior to the external condyle, being luxated posteriorly. At the shoulder abduction of the humerus is possible through an arc of 60 degrees, flexion through 45 degrees, and extension through 20 degrees. External rotation at the shoulder is possible until the flexed forearm just passes the sagittal plane. Internal rotation when the humerus is abducted is similarly limited just before the forearm reaches the coronal plane. The motions of the scapula are not limited. The head of the humerus is dislocated posteriorly beneath the spine of the scapula. The acromion is prominent, with its upper flat surface looking outward, being twisted through an arc of 45 degrees. The clavicle is subluxated upward. The coracoid is normal. Active movements: he has scarcely any grip in his hand; there is slight power in the lumbricals and interossei, fair power of flexion of the thumb, very little adduction of thumb. The wrist cannot be actively extended (Fig. 83, *B*), but if hyperextended passively it can be held weakly in extension momentarily (Fig. 82). There is no power of rotation in the forearm. The elbow can be actively extended to 145 degrees, and flexed only to 70 degrees (passive flexion possible to 50 degrees). At the shoulder there is active but weak abduction of about 30 degrees, flexion of 30 degrees (secured solely by rotation of the scapula), and no extension, no external rotation, but good internal rotation. The axillary fold muscles are good, and there is fair power in the deltoid. The triceps is good. He cannot get his hand to his mouth, it being impossible for him to raise his arm higher than the plane of his shoulders.

Operation (May 31, 1914). Senn's incision, temporary resection of the acromion. The bicipital groove was found directly in line with the external condyle of the humerus, apparently owing to the gradual internal rotation of the shaft of the humerus below the tuberosities. Section of the tendon of the subscapularis at once allowed external rotation of the arm until the forearm was in the coronal plane. It was still impossible to secure complete reduction of the dislocated head of the humerus, owing to tenseness of the pectoralis major. The tendon of this muscle was then divided through another incision (in the line of the anterior axillary fold), and at once easy reduction of the head was secured by external rotation, abduction, and slight pressure forward on the head. If abduction was carried too far the humerus again luxated posteriorly and upward (tension on teres major?). The most stable position was in abduction of 70 degrees and external rotation about 20 degrees short of the coronal plane. The outer end of the clavicle, which was luxated upward, was excised, and this allowed the acromion to come back into better position; but the tip of the acromion was so far bent downward that it kept the head of the humerus pushed backward. Therefore a wedge-shaped piece (base upward) was cut off the portion of the acromion temporarily displaced (not from the spine of the scapula), so that when the remaining portion of the acromion was replaced against the spine of the scapula, the tip of the acromion no longer interfered with the proper position of the humerus, but rode above it. The acromion was fixed against the scapula with a screw. The muscles and skin were sutured separately with interrupted sutures of chromic gut, and the arm was dressed in plaster of Paris in abduction of about 70 degrees and external rotation almost to the coronal plane (Fig. 82).

First dressing six weeks later, when another cast was applied and worn until three months after operation. It was next decided to operate on the elbow, as pronation and limitation of extension persisted and were evidently due to the dislocation of the head of the radius.

Second Operation (October 14, 1914). Kocher's external incision. The brachioradialis and extensor carpi radialis were detached from the supracondylar ridge and pushed forward, and the anterior surface of the capitellum was exposed. The external lateral ligament was then cleared and the head of the radius was exposed in front of it. The head luxated posteriorly in extension of the elbow and limited further extension. The neck of the radius was divided by a Gigli wire saw, and the head removed. This allowed 20 degrees more of extension. The anterior capsule of the elbow-joint was next divided, and then the anterior tendinous surface of the brachialis anticus, its muscle fibers readily yielding to extension when once the aponeurosis was divided. Complete extension was not even yet possible, so the olecranon was exposed behind the external lateral ligament, by displacing the triceps backward, and its tip was cut off (1.25 cm.) by osteotome. Extension to 170 degrees was now possible. The wound was closed in layers, and the arm dressed on an internal angular splint.

November 30, 1914. Passive motion in elbow 65 to 145 degrees. The wrist is no stronger than at first examination, but he now has a fair grip in his hand. He cannot flex his elbow actively beyond 90 degrees, and cannot get his hand to his mouth.

December 7, 1914. Brace applied to overcome wrist-drop.

January 25, 1915. Elbow, passive movements, 65 to 155 degrees; active, 85 to 155 degrees. Shoulder movements are more free than before operation, and chief disability now is from wrist-drop.

Third Operation (March 3, 1915). Curved incision over extensor surface of wrist, slightly convex to ulnar side, from base of index metacarpal to 3 inches above wrist-joint. Annular ligament incised between thumb and index extensors, opening wrist-joint. Radius bared of periosteum. With twin circular saw a slot (6 to 7 mm. in width) was cut in extensor surface of radius, across carpus (scaphoid, trapezoid, and os magnum) and in adjoining surfaces of index and middle metacarpal bones. The slot was cleared of bone with chisel and gouge forceps; all fragments were preserved. A transplant of corresponding width and length was then cut from the subcutaneous surface of left tibia, and inserted in the groove at the wrist. Six transverse saw cuts were made in the transplant so as to allow it to be bent to permit slight hyperextension of the wrist. It was fixed in place by suturing periosteum and fascia over it, with No. 3 chromic gut. The fragments removed from the wrist were then inserted in the defect in the tibia, and both wounds closed. The wrist was dressed on a palmar splint of gypsum in slight hyperextension.

March 15, 1915. Returns to Dispensary for dressing.

March 22, 1915. Good union in wrist. Incision healed.

April 12, 1915. Skiagraphs show transplant in good position, and leg filling in with fragments transplanted from wrist. Gypsum splint continued.

May 24. Binder's board splint applied. Wrist has lost its hyperextension, but remains at 180 degrees.

June 28. Carries objects in hand, dresses himself, and can hold his French horn better. (He plays in an orchestra.)

July 26. Can now close all fingers but fifth. Improvement is marked.

October 5. A few days ago he broke his transplant in the wrist, while asleep, in turning in bed. This allows motion in the wrist of 30 degrees (180 to 150 degrees). Hand continues to grow more useful.

October 14, 1915. Skiagraph shows transplant incorporated with radius and ankylosed to the index metacarpal.

March 12, 1917. Nearly three years after first coming under observation: He finds his hand perfectly useful in playing the French horn. (It should be mentioned that the French horn is played by the left hand, and that the right hand is placed in the flaring end of the horn to support its weight, etc.) He has practically normal use of the thumb, index and middle fingers. The fourth and fifth fingers have very little power. There is passive motion at the wrist from 170 to 140 degrees, flexion being possible actively, but there being no active power of extension (Fig. 83). Pronation in the forearm is complete, both passively and actively; supination, actively and passively, is possible to the midposition; that is to say rotation has increased from a range of 30 degrees when first seen to a range of 50 degrees or more at present. There is passive motion at the elbow from 60 to 160 degrees, and active motion from 90 to 160 degrees. At the shoulder passive abduction is 60 degrees, and active 45 degrees; passive flexion is 90 degrees and active flexion 60 degrees. He is still unable to get his hand to his mouth or to the small of the back.

Compared with his condition when first seen, his present condition is markedly improved; both his parents and himself are satisfied.

CASE XI.—*Disability from contractures; open tenotomies.* Joseph V. was brought to the Episcopal Hospital when twelve years old, April 19, 1915, for disability persisting from an injury to the right shoulder region during birth. He had four sisters, none of them injured in birth, and one brother, who had died of unknown cause (no birth injury).

Examination. He carries his arm in internal rotation, and he cannot put his hand to his mouth nor behind his head. He cannot flex his elbow beyond 90 degrees nor can he fully extend it. Rotation in the forearm (active and passive) is limited in both supination and pronation. Passive movements in the elbow and wrist are not

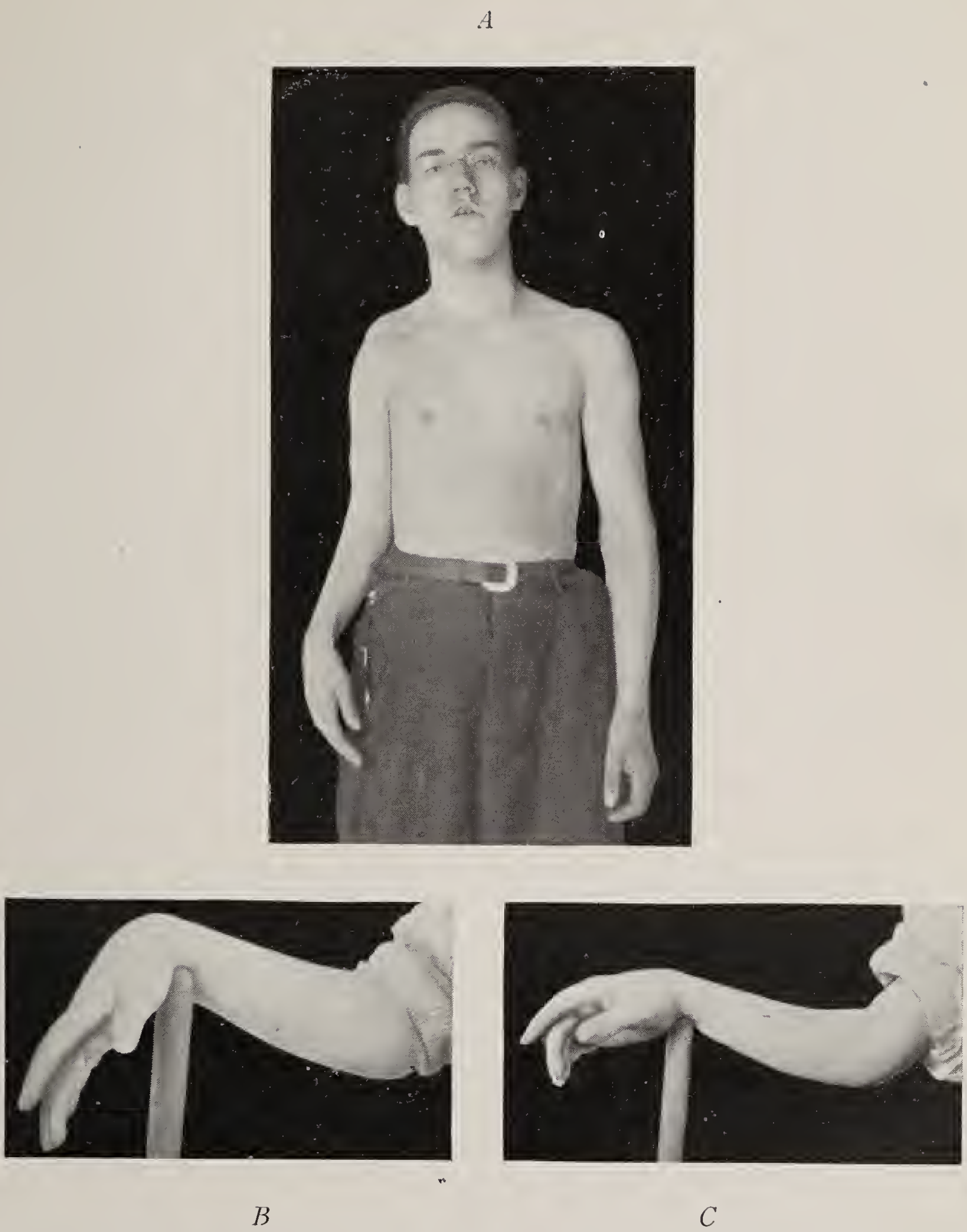


FIG. 83.—Case X. *A*, photograph made September 21, 1914, four months after operation on shoulder; external rotation restored (compare with Fig. 82). *B* (February 15, 1915) and *C* (March 3, 1916), hand and wrist before and after arthrodesis.

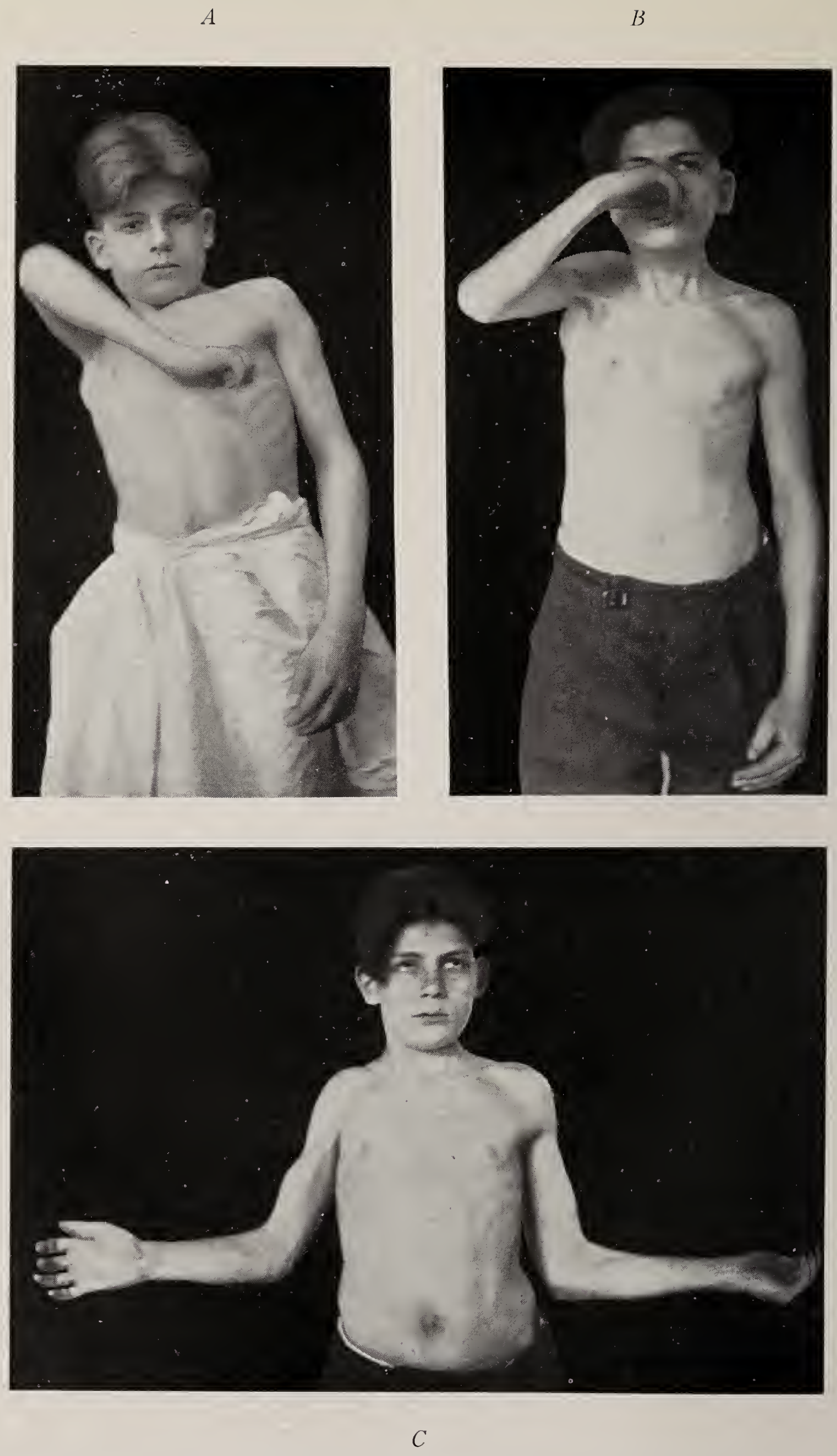
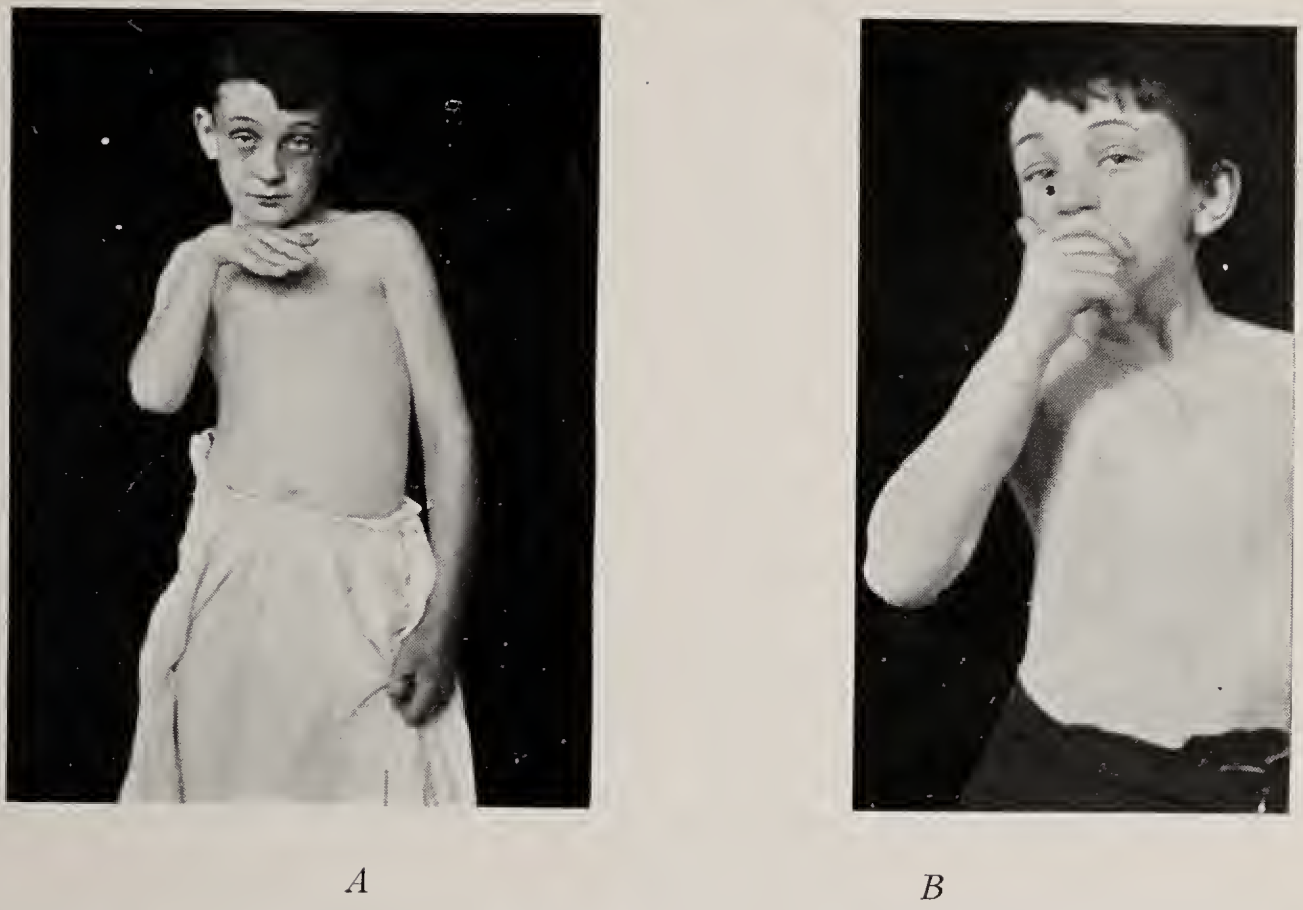


Fig. 84.—Case XI. Showing marked improvement in function secured by tenotomies. *A*, March 20, 1916; *B*, one year after operation; *C*, March 14, 1917, one year after operation.



A B

FIG. 85.—Case XIII. Brachial birth palsy of right upper extremity, with flail shoulder, and loss of supination in forearm and of extension in wrist. *A* disability before operation; *B*, result four months after operations.

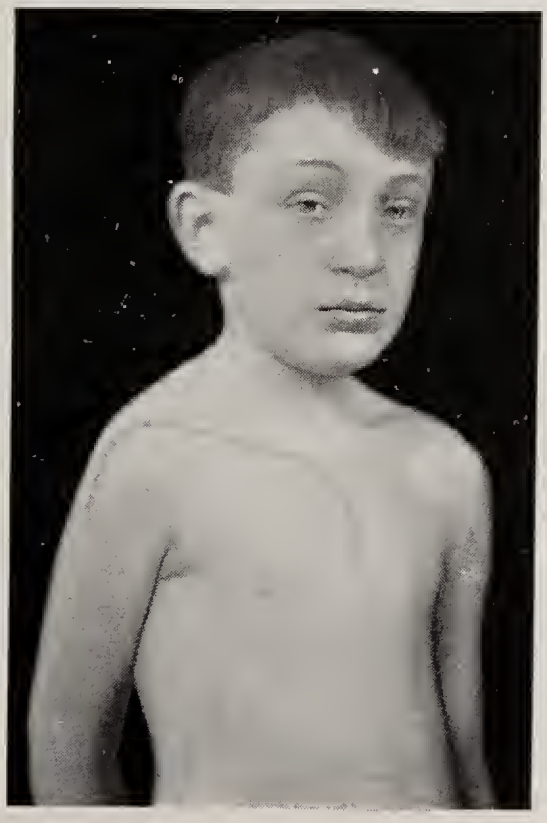


FIG. 86.—Case XIII. Showing the incision employed for transplantation of pectoralis major to take the place of the paralyzed deltoid. Before this operation the humerus became subluxated anteriorly when the position shown in the photograph was assumed, and the arm could not be drawn forward without the aid of the left hand. Voluntary flexion of the shoulder-joint is now easy.

limited. There is passive external rotation at the shoulder only to the sagittal plane; there is no passive extension of the shoulder; passive abduction is possible almost to 90 degrees, but only with the arm anterior to the coronal plane (in partial flexion). Active movements of the hand and fingers are normal. There is good active flexion of the shoulder (by contraction of the coracobrachialis and pectoralis major), but no active extension nor abduction. There is no posterior dislocation of the humerus. The end of the clavicle is slightly above the level of the acromion.

It was recommended that the limitations to external rotation and abduction at the shoulder be overcome by a series of plaster casts. This was declined by his parents, and the boy was not seen again for almost a year (March 13, 1916). No improvement had occurred, and operation was recommended and accepted (Fig. 84, *A*). Reëxamination shows slight posterior subluxation of the humerus.

Operation (March 20, 1916). Ether. Incision as for excision. Division of tendon of pectoralis major allowed normal abduction. The latissimus dorsi was not tight in abduction. Division of the tendon of the subscapularis, while it was kept under tension by attempts to secure external rotation, allowed suddenly full external rotation. This incision passed through the joint capsule, making the head of the humerus visible, and causing it to tend to luxate anteriorly; but even in full external rotation and abduction it did not actually luxate. The bicipital groove seemed displaced externally, looking outward rather than forward. Wound closed without drainage and arm dressed in full abduction and external rotation in plaster-of-Paris.

March 25. Went home.

May 5. New case applied.

June 9. Case removed. Ordered massage and passive motion.

June 25. Can put cap off and on.

March 7, 1917. One year after operation. Gets hand to mouth easily in supination, but arm still abducts slightly in this movement (Fig. 84, *B*). Uses right hand now in writing (formerly used left). Active external rotation is possible to 60 degrees beyond the sagittal plane (Fig. 84, *C*), passive external rotation perhaps 10 degrees further. Active abduction is possible through an arc of 60 degrees, passive abduction perhaps 10 degrees further.

CASE XII.—*Disability from contractures; open tenotomies.* William L. came to the Episcopal Hospital November 23, 1914, at the age of eleven years. His right arm had been injured in birth, and great disability persisted.

Examination. Carries arm in internal rotation and cannot get hand to mouth; even with the aid of the left hand he can get his right hand to his mouth only with wide abduction of the humerus, and with the hand in full pronation. His hand is practically useless. There is atrophy of the supra- and infraspinatus muscles, of the biceps and triceps; and marked contractures of the pectoralis major, the subscapularis, and to a less disabling degree, of the latissimus dorsi. Passive external rotation is impossible beyond the sagittal plane. Extension of the elbow is limited.

Operation (December 9, 1914). Ether. Incision as for excision of shoulder. Subscapularis was divided at its insertion into the lesser tuberosity; this at once allowed external rotation to the coronal plane. Then the tendon of the pectoralis major was lengthened by the usual method of tendon lengthening, known as Z-plasty, the lower half of the insertion being divided close to the humerus, and the upper half of the tendon being cut about 3 cm. nearer the chest. Thus fully one inch lengthening was secured. Dressed in the usual position (abduction and external rotation) in plaster-of-Paris.

December 15. Went home.

January 22, 1915. Six weeks and a half after operation: case removed.

February 1. Cannot get hand to mouth in supination. Ordered massage and passive motions.

February 8. Learning to write with his right hand (previously used left hand). Can easily put hand to mouth in supination, and to the top of his head. The improvement was most striking.

March 22. Can put hat off and on, and put hand to back of neck

May 24. Very good use of hand. Still tends to abduct humerus in flexing elbow. Passive external rotation of the humerus is as good as on the left.

December 6, 1915. Uses hand for everything. Good grasp. Full extension of the elbow. Active external rotation to the sagittal plane.

January 17, 1916. Almost complete active supination.

February 12, 1917. There is power in the deltoid. Can hold his hand above his head. There has been continued improvement during the year.

Case XIII.—*Flaccid paralysis of shoulder (right) with flail joint; transplantation of pectoralis major to supplant deltoid; of pronator radii teres to become supinator, and of flexors of carpus to become extensors.* Carl C. was thirteen years of age when he was brought to the Episcopal Hospital, October 20, 1913. He is the first child of his parents; the second and fifth children died at birth, but the third and fourth children are normal. This patient was born by instrumental delivery (head presentation), and his right shoulder was injured during the process. He was treated by massage and electricity from the age of seven weeks to that of one year. Since that time nothing had been done, and his parents were now anxious to know if something could not be done to lessen his disability.

Examination. He carries the right arm rotated in, the elbow flexed, and the forearm pronated. The limb is useless. Passive movements were possible as follows: Shoulder can be abducted to 60 degrees; can be rotated externally about half the normal extent; while flexion and extension in the shoulder are normal. Flexion in the elbow is normal, but extension is very slightly limited. Wrist motions are normal. Supination beyond the midposition is impossible. Active motions are as follows: scarcely any motion in shoulder, but the axillary fold muscles are good. The biceps and triceps have fair power. There is no active supination; no power in the extensors of the carpus; extensors of the fingers are good, flexors of the carpus and the fingers are good. He can put the back of his hand to his mouth, but not the fingers, owing to the persistent pronation (Fig. 85, *A*). Whenever the arm gets back of the plane of the body the humerus luxates forward at the shoulder-joint and it remains luxated anteriorly until with his left hand he pulls the paralyzed arm forward into a position of flexion and adduction. This causes great disability, and is evidently due to the complete paralysis of the deltoid. Limitation of external rotation at the shoulder and persistent internal rotation indicate there is no paralysis of the internal rotators.

The treatment proposed was to secure supination of the forearm and to prevent the recurrent anterior luxation of the shoulder.

Operation on Forearm (August 27, 1913). Esmarch band above elbow. The pronator radii teres was transplanted through the interosseous space around the posterior and external surfaces of the radius, and while the forearm was maintained in full supination (which became possible after section of the teres muscle) it was sutured to the lateral and flexor surfaces of the radius, the sutures being passed through drill holes in the radius. Next the flexor carpi radialis was transplanted (superficial to the thumb extensors) into the extensor surface of the base of the index metacarpal. Finally the flexor carpi ulnaris was transplanted to the extensor surface of the carpus (unciform or cuneiform bone). The arm was dressed on an anterior angular splint, with the forearm in full supination and the wrist hyperextended.

September 17. First dressing: all incisions healed. Can carry hand to mouth in supinated position. Wrist stays hyperextended.

Operation on Shoulder (September 24). Incision from third costal cartilage to sternoclavicular joint, along clavicle to acromion, thence down outer surface of shoulder nearly to insertion of deltoid (Fig. 86). This large flap was turned down and the clavicular and upper sternal fibers of the pectoralis major were cut at their origin, and, with care to preserve their nerves and vessels, these portions of the muscle were shifted out over the paralyzed deltoid and were sutured to a groove cut by Hey's saw in the acromion and outer third of the clavicle. The incision was closed without drainage. A continuous linen suture was employed for the skin. The limb was put up in plaster-of-Paris, in abduction and external rotation, with the forearm still in full supination.

October 4. Went home.

October 20. Sutures removed through opening in case. Incision healed.

November 3. Case removed, six weeks after operation. Arm carried in sling. Massage ordered.

November 10. Sling removed. Passive movements ordered.

November 24. Can put hand to mouth in supination well. When arm is put behind back he can pull it forward easily by action of transplanted pectoralis major. He can actively extend the wrist, but there is still a tendency to ulnar deviation of the hand.

December 8, 1913. To go to school. Can take hat off and on with his right hand.

December 15. Slight active supination. Photograph in Fig. 85 shows his present condition.

December 27. Can cut his meat with his right hand.

January 17, 1916 (two years later). Works as messenger boy. Can button coat with right hand; can with difficulty get hand to back of head and to mouth (but not in supination). Wrist is in ulnar deviation. He can actively flex and extend wrist and fingers. Active supination is weak. The biceps is good (formerly very feeble). The head of the humerus is in position. There is no active abduction, but he can carry arm forward in sagittal plane (flexion of shoulder) by contraction of the transplanted pectoralis major, which can be felt contracting well.

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A CASE OF SPRENGEL'S DEFORMITY—CONGENITAL ELEVATION OF THE SCAPULA[1]

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R. T., a girl, aged three years, was born with a congenital elevation of the scapula. There is no history of similar deformity elsewhere in the family (Episcopal Hospital).

Examination on Admission. The left scapula is elevated about 1½ inches, with only ¼ inch motion up and down and with but slight motion on rotation. The upper angle lies in the posterior cervical region 1¼ inches below the mastoid process. The upper angle and the upper margin are distinctly hooked forward. The posterior margin has a marked angulation at its center and forms an angle at this point of almost 90 degrees. From this point of angulation a distinct firm band can be felt extending to the sixth cervical vertebra. The scapula rotates about this angle where the band is attached. The child is unable to elevate her arm above her head, nor can the arm be placed in this position passively. The hand cannot be placed behind the neck.

The operation was performed on October 8, 1919. A curved incision, about three inches in length, was made just behind the posterior border of the left scapula, with the lower end of the incision slightly below the middle of the scapula. Skin and fascia were divided. A plate of bone was found extending from the point of angulation at the middle of the posterior border of the scapula obliquely upward to the sixth cervical vertebra. The scapula in the region of this posterior angulation was cartilaginous. The angle of the scapula was excised with the plate of bone attached. A distinct articulation occurred at this point. It was then found

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[1] Transactions of Philadelphia Academy of Surgery, Nov. 3, 1919. Reprinted from Annals of Surgery, February, 1920.

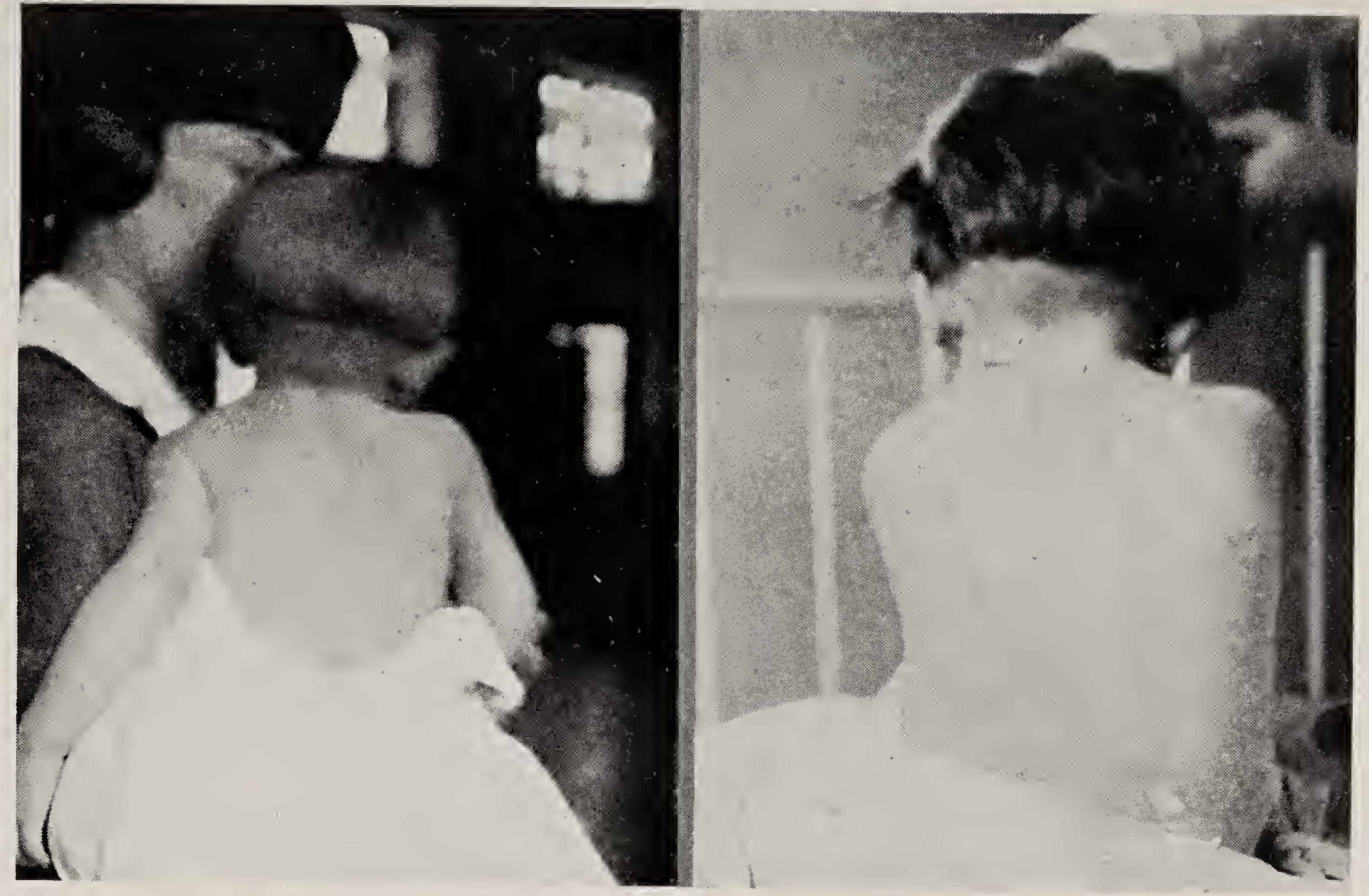

Fig. 87 Fig. 88

Figs. 87 and 88.—Case of congenital elevation of the scapula, before and after operation.

that the bony plate could be moved freely through an articulation with the spine. It was detached from the vertebra.

The scapula could not be brought down to a normal position. The trapezius muscle was separated from the posterior portion of the spine and the two rhomboids and the levator anguli scapulæ were also detached from the scapula. It was found that the supraspinatus was made tense on attempting to move the scapula downward. It was therefore loosened from its posterior attachment. The angle and the upper portion of the scapula as far as the suprascapular notch was found to be sharply hooked forward. This portion of the scapula was excised. The scapula could then be brought down into almost normal position and there seemed to be no tendency for it to return. Wound closed. Child was put to bed on a Bradford frame, with the left hand fastened to the upper part of the bed to maintain the arm in extreme abduction and to rotate the scapula and hold the posterior border down.

November 3, 1919. Wound is healed and the scapula is in almost normal position, the lower angle being but very slightly above the lower angle of the opposite scapula. The child's arm can be held above her head and placed behind her neck. There is free rotation and up-and-down motion of the scapula. Daily active and passive exercises must now be given in order to develop and maintain free motion of the scapula and the upper extremity.

I detached the trapezius from the posterior portion of the spinous process of the scapula and did not notice whether there was any accessory process of the muscle or not. I did not dissect out the muscle, as I wanted to do as little injury to the parts as possible.

The etiology of this deformity as mentioned by Dr. Rugh is interesting. It is known that the upper extremity develops in the cervical region, and during fetal life descends to the position which it occupies at birth. In Sprengel's deformity there has been an arrest of the normal descent of the upper extremity. The cause of its failure to descend is not absolutely clear, but in cases such as the one shown tonight it is possible that the presence of the suprascapular bones will account for it.

SURGERY OF SPASTIC PARALYSIS[1]

By A. BRUCE GILL, M.D.
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Spastic paralysis of infancy and childhood is a common affection, and its treatment has been more or less unsatisfactory. While the orthopedic treatment of anterior poliomyelitis has become quite definite and well established, and is productive of splendid results; the treatment of spastic paralysis has been comparatively neglected, and it remains more or less obscure and unsettled, and its results are uncertain.

Tubby and Jones collected 837 cases, of which 510 were hemiplegic, 157 diplegic, 140 paraplegic, and 30 monoplegic. The paralysis may be of intra-uterine origin, due to cerebral defects, hemorrhage and softening, syphilis, specific fevers, eclampsia and convulsions, etc.; or it may be due to injuries to the head during birth; or, finally, it may be acquired after birth from hemorrhage, chronic meningitis, hydrocephalus, etc. It is common in children of premature birth. Thus, it is seen there is a wide variety of causes of spastic paralysis, and according to the degree of injury or lack of development of the brain or the spinal cord there is a wide divergence in the extent and degree of the spasticity and weakness. Unfortunately, many children with spastic paralysis are mentally subnormal or even idiotic. This renders the prognosis more unfavorable and the treatment more difficult.

I shall not take the time to consider the various forms and

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[1] Reprinted from Annals of Surgery, May, 1918.

the symptoms of this affection. Suffice it to say that they are dependent upon the distribution and degree of spasticity and weakness and upon the mental characteristics.

Nor shall I here discuss what cases are unsuitable and what ones suitable for treatment, nor the indications for and the methods of conservative treatment; but shall speak briefly of the various operative measures that have been employed to improve the condition of these patients.

Tenotomy of the contractured muscles is the oldest and the most commonly employed surgical procedure for the relief of spastic paralysis. This permits the limb to assume a normal position. It has two values: it relieves the weak opposing muscles from constant overstretching, and it breaks the vicious circle formed by tendon, afferent sensory nerves, spinal center, efferent motor nerves, and muscle. If the weak muscles, which are the opponents of the strong contractured muscles, are relieved from the strain of overstretching, they will in a measure recover their strength and be better able to resist the pull of their strong opponents which are temporarily disabled by tenotomy and which will for some time be partially handicapped mechanically by the lengthening which they have suffered. In the second consideration, it would appear that the tension of a muscle is somewhat dependent upon the tension of its tendon. If pull is made upon the tendon of a muscle a message is carried by sensory nerves to the spinal center, whence an efferent impulse is sent out to the muscle which causes it to contract to resist the tension upon its tendon. Normally such impulses are controlled by the cerebral centers, but in spastic paralysis the cerebral centers have been disconnected from the spinal centers, have lost their control, and can no longer inhibit the excitability of the peripheral arc. Therefore the peripheral arc remains in a constant state of oversensitiveness and overexcitability. This is manifested in the contracted muscles and the exaggerated reflexes. The tenotomy of a tendon cuts this arc, or circle, at one point, and breaks the stream of afferent impulses that were constantly pouring in upon the spinal centers. The centers, therefore, quiet down, and on their part cease to send forth the continuous stream of efferent impulses to the muscles that kept them in a state of constant contraction.

Unfortunately, tenotomy of contracted muscle tendons has not proved uniformly successful in practice. In many cases there has been a recurrence of the original condition. In numerous other cases an opposite deformity has been produced. A talipes equinus has been converted, for example, into a talipes calcaneus, which is a more disabling deformity.

Therefore, Foerster proposed and executed his posterior spinal root resection. This operation cuts the vicious circle by dividing the sensory nerves just before they reach the spinal centers. This surgical procedure is, however, so difficult and severe as to preclude its common use.

Stoffel then attacked the problem in a different manner. He pointed to the fact that in spastic paralysis all muscles are alike spastic; and that, for example, in talipes equinus the anterior leg muscles are spastic as well as the posterior muscles, but that the latter are so much stronger they overpower the former. The anterior muscles are overstrained and overstretched and thus become definitely weakened. This weakness is not a real paralysis. In fact, in spastic paralysis there is no absolute paralysis. In the second place, tendons that often appear contractured are not really so. The muscles are shortened only by spastic contraction that disappears under ether. No real muscle shortening is present. Therefore a tenotomy lengthens a muscle which is not shortened and permits the development of an opposite deformity. If contracture persists under a general anesthetic it is permanent, and the tendon should be lengthened: Stoffel therefore proposed to weaken the strong muscles by a surgical procedure and to strengthen the weak muscles by freedom from overstrain and by exercise. Thus he would establish an equilibrium of flexors and extensors, for example, that would maintain the limb in normal position and allow more or less normal function. He weakens the strong contractured muscles by resecting a portion of their motor nerve supply.

To correct talipes equinus the internal popliteal nerve is exposed in the popliteal space. It is dissected into numerous bundles which are found to supply the various muscles of the calf of the leg. A portion of each bundle supplying a spastic contractured muscle is then excised for a length of one to two inches. Approximately one-fourth to two-thirds or three-fourths of the nerve supply to a muscle is thus resected depending on the amount of spasticity and deformity present in each case. The electrode is used in distinguishing the nerve-bundles which supply the various muscles, except when the surgeon's knowledge of the anatomy of the nerves enables him to dispense with its assistance.

For contracture of the hamstring muscles, operation is performed upon the sciatic nerve in the upper part of the thigh. For adductor spasm one or both branches of the obturator nerve are excised. In the upper extremity the median nerve is exposed in the flexure of the elbow and the branch going to the pronator radii teres and the branches to the various flexor muscles of wrist and fingers are resected as desired.

During the last four years the author has performed thirty-five Stoffel operations on the popliteal, the sciatic, the obturator, and the median nerves. A complete report of these cases will be published later. The results have been so satisfactory that the operations have been done routinely in the author's orthopedic service at the Episcopal and the Presbyterian Hospitals and in Dr. Ashhurst's service at the Orthopædic Hospital.

The following cases illustrate briefly the results that have been obtained:

Case I.—C. C. Spastic paraplegia since birth. September 14, 1914. Is able to walk with the greatest difficulty. September 26, 1914: Operation upon both obturator and both internal popliteal nerves. April 25, 1915: Able to walk fairly well, heels come to the floor but toes strike the ground first. October 19, 1915: Second operation upon both popliteal nerves. January 18, 1916: Heels on the floor in walking; walks much better

Case II.—E. M. Spastic hemiplegia. July 20, 1915: Marked flexion of right wrist and fingers. Foot in marked equinus; walks on toes. July 21, 1915: Operation on right popliteal nerve. At the same time the tendo Achillis was tenotomized and the extensor proprius hallucis was transplanted to the head of the first metatarsal. All the flexor tendons of the right wrist were lengthened. December 1, 1917: Walks well, with but slight limp; foot is straight and firm on the floor in walking; right hand is markedly flexed at the wrist and the fingers partially flexed in the palm. A Stoffel operation will now be performed on the median nerve as the tendon lengthening has not cured her deformity.

Case III.—W. G. November 1, 1914: Left arm: forearm pronated; wrist flexed; fingers and thumb flexed into palm; no active extension of wrist; slight motion of finger but none of thumb, except slight adduction and flexion. Left leg: walks on toes; no active motion in ankle; tendo Achillis contractured; hollow-foot; hammer-toe. November 4, 1914: Operation on median and internal popliteal nerves; tenotomy of tendo Achillis and transplantation of extensor proprius hallucis to the first metatarsal. December 7, 1914: Wrist, fingers and thumb are straight. May 10, 1915: Can actively extend fingers almost to normal limit; can make good fist; can move wrist feebly. No active supination. September 13, 1915: Good grip; slight control of thumb; can hold objects in his hand.

Case IV.—R. J. February 26, 1914: Walks on toes, with legs crossed; very unstable. October 2, 1914: Operation on both obturators and internal popliteal. January 9, 1915: Much improved; heels on ground when walking; legs straight; does not drag toes; walks with braces.

Case V.—C. J. March 20, 1915: Contracture of tendo Achillis; ankle and patellar clonus. March 24, 1915: Operation; partial resection of nerve supply to gastrocnemius, soleus and peronei. May 1, 1915: Foot in good position. Can dorsiflex foot well. Much improved in walking.

These cases, which are but a few of those operated upon, illustrate the improvement which occurs after nerve resection. In several instances a second operation was performed, when it was found that too little of the nerve supply had been resected at the first operation. It is better to resect too little than too much. In operation upon the lower extremity the results have appeared to be almost uniformly successful and satisfactory. In resections of the median nerve of the arm the results are not functionally so good, although the cosmetic results are satisfactory.

Following operation the after-treatment should be thorough and persistent in educating the weakened muscles and in securing coördination. Unfortunately, the mental condition of the patients and the lack of proper facilities for the work interfere greatly with the training. Notwithstanding these difficulties, the Stoffel operation appears to the writer to afford the best solution yet offered for these cases.

It may be mentioned that tendon transplantations, especially in the arm, may be productive of more or less improvement. Nutt's operation of intraperineural neurotomy with immediate re-suture has also been performed with success.

The writer has considered a modification of the Stoffel operation—instead of partial nerve resection, he proposes a transplantation of the same nerves into the weak opposing muscles. Anatomical difficulties would not permit of the universal application of this procedure.

It would be interesting to know what occurs in the muscles following the Stoffel operation. Part of the nerve supply of the muscle has been removed and a portion of the muscle paralyzed. It seems probable that in time the remaining nerves will neurotize the entire muscle. Only time will tell whether the good results of the operation will be permanent.

Sharpe's cerebral decompression for spastic paralysis is on trial, but it would appear to be of value only in recent cases, and particularly in the newborn.

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DEFORMITIES OF THE HAND[1]

By A. BRUCE GILL, M.D.
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In preparing this paper for the benefit of the general practitioner, one is confronted by the difficulty of presenting a large subject which is of interest, for the most part, only to the specialist. It seemed best, therefore, to enumerate briefly the various deformities to which the hand is subject and then to dwell more fully on one or two conditions which are of particular interest to the general practitioner.

A deformity of the hand may be either congenital or acquired. The axis of the hand normally is the continuation of the axis of the forearm. If there is any change in the relations of these two axes a condition of club-hand results. Congenital club-hand may be due, therefore, to, first, a partial or complete defect in the bones of the forearm, or, second, a condition analogous to club foot, *i. e.*, there is no bony defect. The latter is rare. The former is fairly common. Congenital deformities of the fingers include contracture of the fingers, syndactylism, or webbed fingers, polydactylism, or supernumerary fingers, partial or complete suppression of the fingers, and hypertrophy of the fingers.

In cases of syndactylism, in which the webbing extends all the way to the nails, operations may not be entirely satisfactory. The best results are secured by plastic operation, in which the skin from the dorsum of one finger is used to cover entirely the skin defect in the other finger, and in which a flap from the dorsum of the hand is employed to cover the raw surface of the first finger. The difficulty is, sometimes there is sloughing of the flap, or the flap may not have been large enough to extend to the tip of the finger, or if the flap has been large enough to cover the entire raw area, and no sloughing has resulted, even then contracture occurs, along the line of the suture, and the finger becomes curved to one side. Secondary operation may be attempted later to remove scar tissue and to transpose another pedicled flap from the dorsum of the hand.

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[1] Reprinted from New York Medical Journal, December 27, 1919.

Polydactylism is a fairly common condition and is usually hereditary. The supernumerary digit should be amputated. In deciding which is the supernumerary digit the surgeon should be guided by the *x*-ray, which will indicate which finger is the direct extension of the metacarpal bone. Sometimes there may be a supernumerary metacarpal or a metacarpal may be branched. In such cases the supernumerary bone should be excised.

The acquired deformities of the hand comprise the various forms of club-hand and deformities of the fingers.

Club-hand may be due to:

1. Curvature of the bones of the forearm from disease or from fracture.

2. Defect in the bones of the forearm. Osteomyelitis may destroy a portion of the shaft of one of the bones of the forearm and thus cause a comparative shortening of the bone. More commonly the lower epiphysis is destroyed, the bone ceases to grow equally with its fellow, if, as is usually the case, the patient is a child, and soon a marked and serious lateral deviation of the hand occurs. The treatment of this condition presents great difficulty because the child has many years of development ahead of him. If the long bone should be shortened by the excision of a portion of its shaft, or if the defective bone should be lengthened by a bone transplant at its lower end, the deformity of the hand might be temporarily

corrected; but it would soon recur as the normal bone continued to outstrip the diseased bone in growth. Should the surgeon defer operation until the patient reaches maturity, the deformity may have become so obstinate that it could not be corrected by any procedure. A deliberate destruction of the growing epiphysis would possibly solve the difficulty, but the surgeon hesitates at so radical a measure. The transplantation of the upper end of the fibula with its epiphysis might be attempted. It is apparent that in any case the affected arm must become shorter than the other arm.

It is possible for a fracture through the lower epiphysis of a bone to destroy the growth of the epiphysis and produce a similar condition of club-hand.

3. Madelung's deformity of the wrist. This consists in a spontaneous backward dislocation of the ulna which does not seem to be caused by any specific disease. It is said to occur chiefly in washerwomen and in pianists. There is a loosening of the ligaments of the lower radio-ulnar articulation, and a progressive displacement of the ulna backward. If seen early some form of support may be sufficient to prevent the progress of the condition; if it should not, operation may be performed to plicate the ligaments, or to secure a bony or fibrous union between the radius and the ulna.

4. Destructive disease of the bones of the wrist and the carpus. Osteomyelitis and tuberculosis of the carpal bones are not uncommon and they produce serious disability of the hand which is out of proportion to the amount of deformity present.

5. Certain paralytic and muscular conditions. Spastic paralysis, infantile paralysis, Volkmann's contracture, which will be discussed later, may produce various types of deviation of the hand at the wrist.

Deformities of the finger may be classified as follows:

1. Contractures which are of cicatricial origin, involving the skin only. These are due usually to burns. I have seen the deformity so severe that all of the fingers were flexed tightly

into the palm, and were bound fast to the very tips. Fairly good results can be obtained by plastic operations. In the severe forms it may be well to amputate one or two of the fingers and utilize the sound skin from them to cover the defect in the palm. Usually the surgeon has to resort to the pedicled skin graft.

2. Contractures of fascial origin—Dupuytren's contracture. I believe the best result is to be secured by a complete excision of the palmar fascia through the distal transverse crease of the palm and the insertion of a free fat transplant beneath the skin.[1]

3. Deformities of the fingers due to affections of the tendons: first, cicatricial contractures from infections or injuries; second, interference with the movement of a tendon within its sheath, producing a condition known as snap, jerk, or trigger finger; and, third, a partial or complete traumatic avulsion of an extensor tendon from its insertion into the distal phalanx, known as mallet finger. This is seen chiefly in baseball players.

4. Contracture of the fingers resulting from lesions which have destroyed more or less of the muscle substance of the flexors or extensors of the fingers. Infections of the forearm and Volkmann's ischemic paralysis illustrate this group of cases.

5. Various forms of arthritis, osteo-arthritis and gout.

6. Nervous disorders, such as spastic paralysis, infantile paralysis, obstetrical, or birth palsy, the definite nerve palsies, such as musculospiral and ulnar, and certain forms of muscular dystrophies.

7. Contractures arising from such diseases of the bones as syphilis, tuberculosis and tumors.

Of all these many kinds of deformities of the hand, I believe that the two which are of the greatest practical interest to the general practitioner are those which are due to Volkmann's contracture and to malunion of Colles's fracture.

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[1] Gill: Dupuytren's Contracture, with a Description of a Method of Operation, Ann. Surg., August, 1919.

Volkmann's contracture usually results from a fracture at the elbow or at the wrist. It occurs within the first twenty-four hours, even within the first two or three hours after the fracture is dressed for the first time, and is due to interference with the blood supply to the muscles of the forearm. The best teaching is to dress a fracture at the elbow in acute flexion of the elbow, the so-called Jones's position. Occasionally the attending physician, whether he be a general practitioner, a hospital interne, or a surgeon does not secure full reduction of the lower fragment or fragments of the humerus. These fragments may be displaced forward. If the elbow is flexed before complete reduction has been obtained, these fragments make pressure in the flexure of the elbow and prevent complete flexion. The attending physician forces the flexion, and thereby compresses the vessels at the elbow, and shuts off, in part or in whole, the blood supply to the forearm. If the fracture is first reduced it is found that complete flexion of the elbow is easy, and does not require force, and the blood supply is not interfered with. In other words, the arm should not be dressed in flexion until the fracture has been fully reduced. Difficulty in securing complete flexion is an indication that this reduction has not yet been accomplished. Dressing the arm in flexion will maintain reduction of the fracture, but it will not accomplish it.

Of course, it is possible for a tight bandage so to constrict the circulation as to cause Volkmann's contracture. When the circulation to the muscles is interfered with, the muscles suffer a consequent degeneration, in whole or in part, and are replaced by fibrous tissue. The muscles, with the tendons, become contractured, and the hand is flexed at the wrist, and the fingers and thumb are flexed into the palm. Not infrequently there is disturbance of sensation of the hand and forearm, even to complete anesthesia in the distribution of a particular nerve. If the condition is not cured, the forearm and hand do not develop *pari passu* with the other hand and forearm. I have seen a shortening of three inches in the forearm of a child twelve years of age, due to Volkmann's contracture. The cure of this condition is extremely difficult. The best results in the severe cases are to be obtained by complete dissection of the muscles involved, whereby they are freed from one another and from the overlying tissues. This dissection should be followed by implantation of a free fat transplant to prevent re-adhesion.[1] Not infrequently the tendons at the wrist require lengthening. Therefore, emphasis is placed on the fact that Volkmann's contracture is a severe and permanent deformity, that it occasions partial or complete loss of function of the hand, which is one of the most useful members of the body; and that this condition is due to improper treatment of a fracture at the elbow, or occasionally a fracture in the forearm; and that it occurs usually at the first dressing. This first dressing is frequently made by the general practitioner.

Another almost equally disabling deformity of the hand may follow Colles's fracture. It is well known that many persons who have suffered a Colles's fracture are unable to resume active work for many months or even years. The chief cause of the disability is the stiffness of the hand and the fingers, which prevents the grasping of objects firmly or even at all. This condition of immobility in the joints develops within the first few weeks, and may persist indefinitely despite treatment by baking, massage and manipulation. The stiffness of the fingers is accompanied by more or less swelling of the fingers and the hand, by glossiness, blue discoloration and coldness of the skin. It is not infrequent to find rather marked pitting on the dorsum of the hand three months after the fracture. What is the cause of this deformity and disability? It is due chiefly to interference with the blood supply to the hand. While some of the limitation of the motion of the fingers may be due at times to fixation of the tendons at the wrist in the callus at the site of the fracture, yet it has often been apparent during operation when the tendons have been freed from all adhesions, that there persists a rigidity in the joints of the fingers and the hand which is the main cause of the disability, and which cannot be overcome even by the use of considerable force. The resistance to motion is not in the tendons of the fingers but in the intimate structure of the joints. In other words, a condition of arthritis and fibrous ankylosis has developed in the hand. Physiotherapy continued through many months will frequently fail to secure good function. I have found, however, that after operation, whereby the deformity at the point of fracture is reduced, or whereby the soft tissues are freed from adhesion to the bone, the return of function in the hand, while delayed, is yet much more rapid than it was preceding the operation. Therefore, the condition which results in the hand following a Colles's fracture may be likened somewhat to the condition in Volkmann's contracture. The malposition of the fragments may indeed interfere with the normal function of the tendons as they pass in close relation to the bones at the wrist; but what is much more important, the malposition interferes with the blood supply to the hand. A condition of congestion of the bloodvessels results, swelling and edema occur, plastic material forms about the joints and produces a fibrous ankylosis. If the fracture were completely reduced and the splint properly applied, the blood supply would not be interfered with, what little swelling of the hand had already occurred or should occur within the first twenty-four hours would speedily subside and it would be found that the patient, within a few days after the fracture would have fair or even unrestricted motion of the fingers. If swelling of the fingers or hand persists for a week after the fracture or at least is not rapidly improving within the week, I think we may consider that the fracture has not yet been sufficiently reduced. It certainly is evident that something has interfered with the normal circulation. I believe this condition of the fingers and hand is the best test we have of knowing whether or not the fracture has been sufficiently reduced. In some patients the x-ray plates show fairly good reduction of the fracture and yet the swelling and disability of the hand continue. In other patients the x-ray plates show more or less marked displacement of the lower fragment and yet the function of the hand is good. It is impossible by x-ray examination to tell absolutely whether or not a good result will be obtained.

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[1] Gill: Plastic (Reconstructive) Surgery of the Hand and the Forearm, Ann. Surg., July, 1918.

What, then, may be done to prevent or to remedy this condition which may result in long-continued disability of the hand and even in inability of the patient to earn a livelihood? If the fracture were completely reduced, the condition would not arise. Reduction is more readily and certainly obtained under a general anesthetic. But even under an anesthetic absolute reduction is not always to be secured. Shall the surgeon then resort to open operation? This question must be answered in the light of what has been stated above. Absolute reduction is not always essential to restoration of normal function, nor, indeed, in Colles's fracture is it commonly obtained. Comparatively few such fractures are completely and absolutely reduced, and yet the majority of patients secure good function of the hand within a reasonable length of time. If, however, the swelling and other signs of interference of the circulation persist beyond the first week, or do not show signs of rapid subsidence at the end of that time, I believe we should conclude that there is something still interfering with the circulation at the wrist and that the surgeon should then employ open operation.

One hears much discussion as to the sort of splint that should be used in Colles's fracture, and I fear that in our medical schools and hospitals too much attention is given to the form of splint and too little to the reduction of the fracture or to the observation of the conditions mentioned above. No complicated splint is necessary. If the fracture is reduced it is rare for the bones to become re-displaced except by violence. A well padded straight posterior splint is one of the simplest and most efficient means for maintaining reduction. Students are sometimes taught to place anterior and posterior pads at the wrist. These pads will not secure reduction if it has not already been accomplished, and I believe that they are not necessary to the maintenance of reduction and they are only liable by pressure to interfere with the blood supply. I should not advise their use.

The hand should be removed daily from the splint, and a gentle massage, which consists of stroking movements only, can be given to aid in the return of normal circulation. At the end of ten days to two weeks massage should be more thorough and should include the hand and the forearm. I see no objection to allowing the patient to move the fingers within a few days after the fracture; and the splint can be applied only as far as the metacarpophalangeal joints, in order to allow motion in the fingers. Undoubtedly both active and passive motion in a member assists the circulation.

I have dwelt at length on these two conditions of Volkmann's contracture and Colles's fracture, because fractures at the elbow and the wrist are among the most common of all fractures, and because it is unfortunately true that many patients are disabled thereby for much longer periods than they should be, or are even permanently prevented from pursuing their vocation in life.

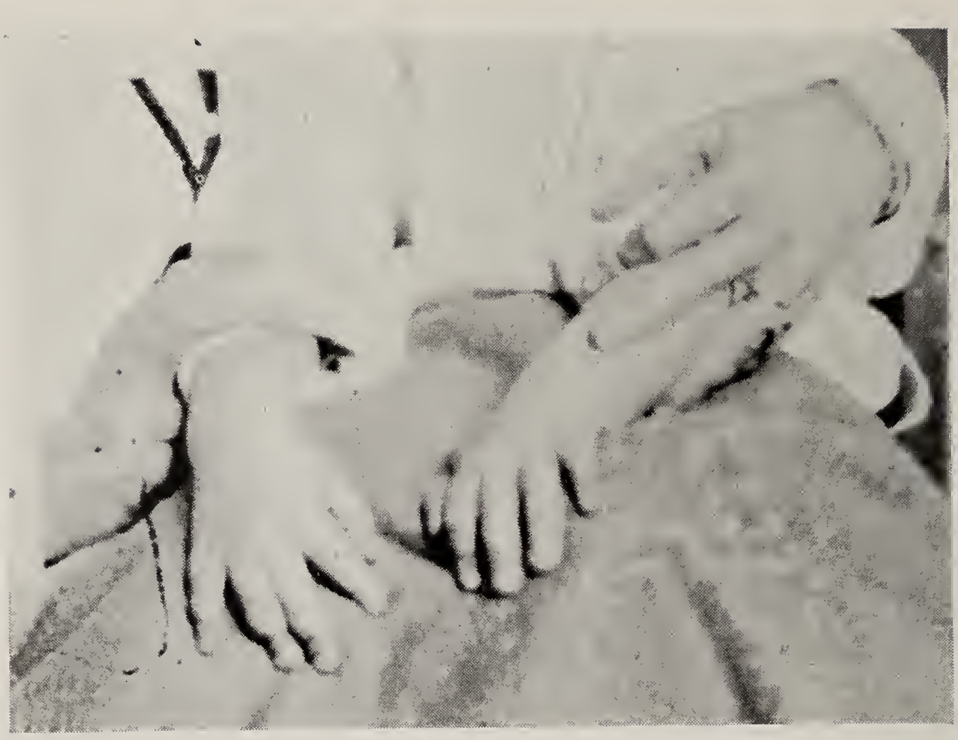

FIG. 89.—Multiple enchondromata.

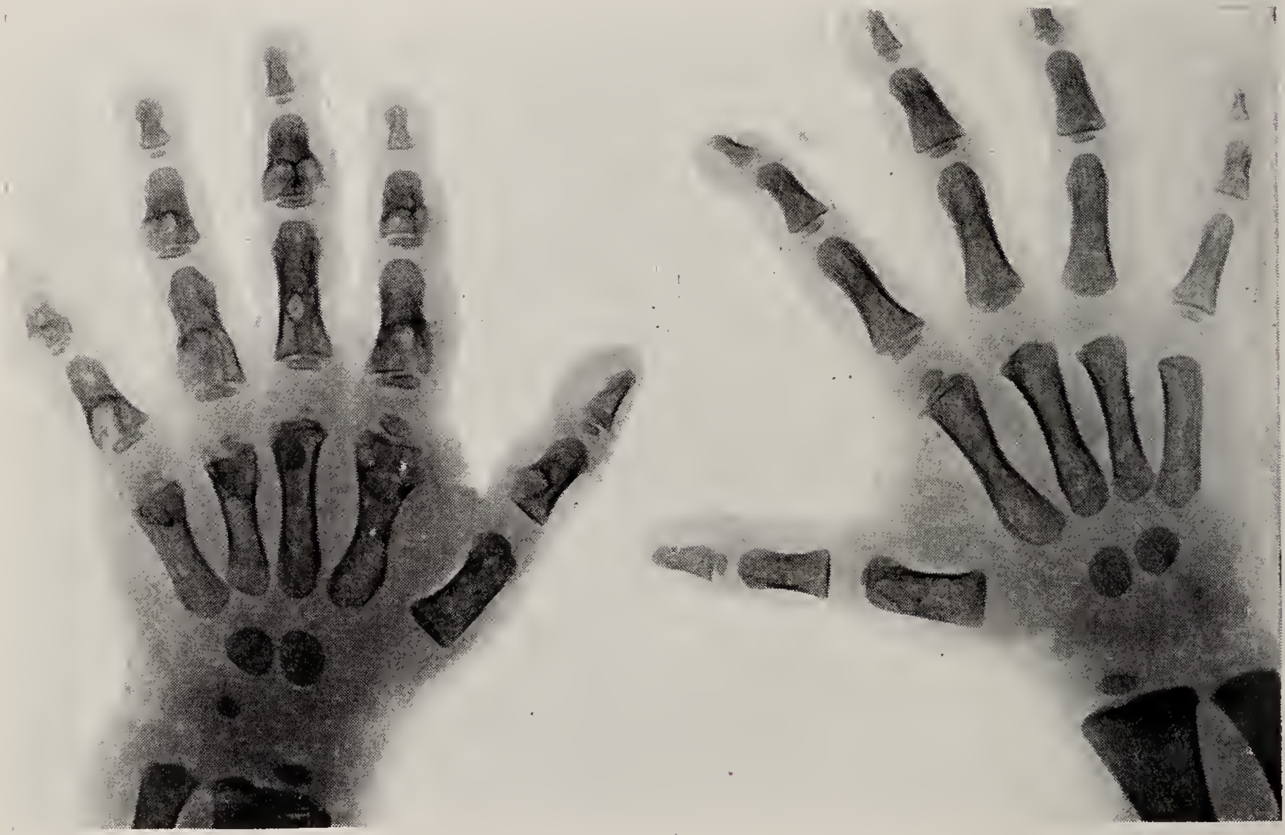

FIG. 90.—Skiagraphs of a case of multiple enchondromata.

MULTIPLE ENCHONDROMATA OF THE BONES OF THE RIGHT HAND OF A CHILD TWO AND A HALF YEARS OF AGE[1]

By A. BRUCE GILL, M.D.
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Dr. A. Bruce Gill presented the case of a male child, aged two and a half years, whose *x*-ray photographs showed multiple enchondromata of the right hand only. Four metacarpals and nine phalanges are involved. *X*-ray plates of the other bones of the body show no evidence of enchondromata.

The child is healthy and well developed. He has never been ill, with the exception of a furunculosis about a year ago. His parents are healthy, and there is no family history of enchondromata or hereditary deforming chondrodysplasia, as Ehrenfried terms it, for a period of at least three generations.

The child's parents first observed three or four months ago that his right hand seemed fatter, as they thought it, than it had been before. He has never complained of any pain or any discomfort in the hand, and he uses it for all ordinary purposes. Examination showed demonstrable enlargement of several of the phalanges and the metacarpals. (Figs. 89 and 90.)

The subject of multiple enchrondromata or hereditary deforming chondrodysplasia, together with its literature, has been presented well in recent years by Boggs,[2] and by such others as Oberndorf, Clark and Atwood, Frangenheim,

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[1] Transactions of Philadelphia Academy of Surgery, May, 1917. Reprinted from Annals of Surgery, November, 1917.

[2] Tr. Assn. Am. Phys., Philadelphia, 1913, xxviii. Ehrenfried: Jour. Am. Med. Assn., May 15, 1915. Ashhurst: Ann. Surg. 1916, lxi, 167.

Schmidt, Lubarsch, Carman and Fisher and others referred to in the above papers.

Various theories as to its etiology have been advanced, and many diseases, such as syphilis, tuberculosis and other infections, rachitis, thyroid deficiency, trophic changes in the central nervous system, have been suggested as causes. But the best opinion appears to be that it is a congenital condition due to abnormal *anlage* in the intermediary cartilage of the bones involved, and that frequently heredity plays a part. Certain cases said by Boggs to have been reported in the newborn, and the case here presented in a child two and a half years of age, strongly indicate that the condition is truly a congenital one. The hereditary influence cannot always be traced, nor is the underlying cause of the congenital abnormality apparent.

DUPUYTREN'S CONTRACTURE, WITH A DESCRIPTION OF A METHOD OF OPERATION[1]

By A. BRUCE GILL, M.D.
ORTHOPEDIC SURGEON TO THE HOSPITAL

THE treatment of Dupuytren's contracture has long been tedious, difficult, and often unsuccessful. Even when temporary restoration of the hand has been secured, relapse has been common.

The author's experience, limited though it has been, in plastic surgery of the hand and forearm, has proved to him the great value of the free fat transplant in preventing adhesions after the excision of scar tissue and after extensive dissections. It has also demonstrated the advantage of making incisions along the line of the natural creases of the hand and fingers in order to secure healing of the wound without danger of subsequent keloid growth, contracture, and adhesion of the scar to underlying structures.[2]

This led the author to employ the following method of operation for Dupuytren's contracture:

1. Under general anesthesia, without the use of a tourniquet, a transverse incision is made along the distal palmar crease. Through this incision alone a careful dissection is made of the entire palmar fascia to or beyond the crease at the base of the thenar eminence, as far toward the base of the palm as is necessary, and to the web of the fingers. The skin is adherent

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[1] Reprinted from Annals of Surgery, August, 1919.

[2] Plastic Reconstructive Surgery of the Head and Forearm, Ann. Surg., July, 1918.

to the fascia, and the dissection must be made carefully to avoid buttonholing the skin. With proper retraction as the dissection proceeds it is found that the entire fascia can be exposed and freed within the limits mentioned.

2. The contractured fascia is then excised without injury to the underlying tendons, vessels, and nerves. The tendons do not require lengthening as they do not participate in the contracture.

If contractured fascia is present on the palmar aspect of the proximal phalanges, it may be excised through transverse incisions along the crease at the base of each finger involved.

If now it is found that the proximal interphalangeal joint cannot be extended, or that it can be extended and flexed again only with a snap, the head of the first phalanx must be excised through a transverse dorsal incision over the joint. Professor J. Hutchinson lays great stress upon the necessity of this procedure in long-standing cases. This is, of course, a familiar procedure to surgeons who are accustomed to operate on hammer-toes or on fingers long held flexed by other causes than Dupuytren's contracture.

3. A small free fat transplant from the thigh is inserted smoothly beneath the palmar skin. It will lie in position without sutures. It is placed here to prevent subsequent adhesion of the skin to the tendons and to reconstruct the normal softness and plumpness of the palm of the hand. The incision is closed with a few interrupted sutures of No. 0 chromicized catgut. The hand is dressed on a well padded splint for a week. Slight serous discharge may occur for a short time, which is probably due to some dissolution of the fat transplant. Infection from the outside may be prevented by the use of dichloramine-T on the wound at the daily dressings. Great care should be taken, of course, at the time of operation to avoid infection.

The following case will illustrate this method of operation:

Case.—J. C., a tool-cutter, was seen at the Episcopal Hospital on July 29, 1918. About the middle of June his right hand was

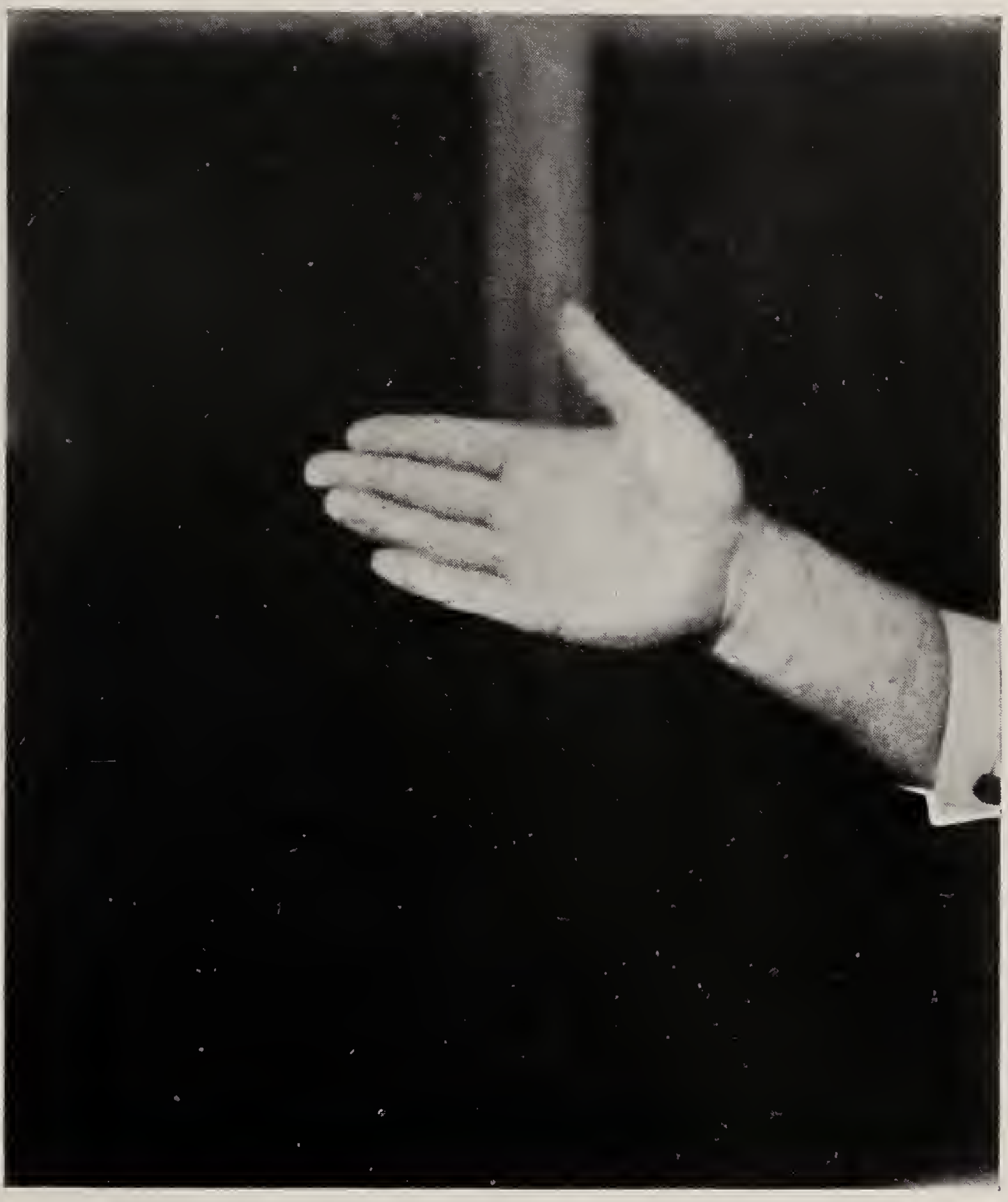

FIG. 91.—Hand of patient three months after operation, showing normal extension of fingers and absence of perceptible scar.

injured in a machine. The palm was contused, but the skin was not broken. The injury was not sufficient to cause him to stop work. Thereafter he experienced soreness in the palm, particularly after working.

On examination there was present a marked thickening on the ulnar side which extended to the middle of the palm of the right hand. Active and passive extension of the little finger was limited to 160 degrees. Massage was ordered.

On August 12 it was observed that the condition had spread since the first examination and that the little finger was flexed more acutely and that the ring finger was also partially contractured.

Later, on September 9, the contracture involved the palmar fascia as far as the base of the thenar eminence, and the contracture of the little and ring fingers was more marked.

Operation was performed on September 23, 1918, by the method described above. The entire palmar fascia was excised from its ulnar border to slightly beyond the crease at the base of the thenar eminence and from the base of the palm to the web of the fingers. It was unnecessary in this case to go beyond the web. A free fat transplant from the thigh was inserted beneath the skin, and the transverse incision was closed with a few interrupted chromicized catgut sutures. The hand was dressed on a palmar splint.

The wound healed, but ten days after the operation its outer portion broke down and discharged a small amount of serum for several weeks. No pus was present. It was dressed with dichloramine-T. Eventually the wound healed again, with a scar that is scarcely perceptible, with no induration in the palm or about the scar, and with normal motion of the finger.

The condition of the patient at the present time is as follows: The scar along the distal transverse crease can with difficulty be seen; the palm is soft and plump, but not unduly so, and the motion and function of the fingers are absolutely normal. The little finger of his left hand has been amputated. A little over a year ago he developed a Dupuytren's contracture of his left hand for which he underwent seven operations in another hospital and eventually had the little finger amputated.

Dupuytren's contracture was described by Sir Astley Cooper, but it was first dissected by Dupuytren in 1831, who demonstrated that the condition is one of contracture of the palmar fascia alone without involvement of the flexor tendons. Sir Robert Jones defines it as a fibrositis of the palmar fascia not associated with any inflammatory thickening of the skin.

Pathologically there is a thickening and contracture of the digital processes of the palmar fascia primarily and of the main body of the fascia secondarily. In extreme cases the interphalangeal joints may become partially dislocated. Microscopically the fascia is found to consist of fibrous bands with cellular infiltration characteristic of a plastic inflammation.

The palmar skin is puckered and usually is closely adherent to the fascia.

There have been two theories as to the etiology:

1. That the contracture is due to external agencies such as acute injury to the palm or oft-repeated stresses. 2. That it is constitutional in origin. Keen, Adams, and Anderson, three leading writers on the subject, lay more stress on the second theory. Sir Robert Jones thinks the cause is probably a predisposition in an individual with palmar irritation as an exciting cause. Kenneth Black, to whose comprehensive article in the *British Medical Journal*, 1915, i, 326, the reader is referred, states that he believes it will eventually be recognized to be due to a certain internal condition (possibly akin to gout or rheumatism) among persons of advancing age.

The case which I have reported above occurred in a healthy young adult as the result in each instance of injury to the hand. But we cannot deny, of course, that some predisposing internal condition was also present, although he presented no rheumatic or gouty tendencies.

Two methods of treatment of Dupuytren's contracture have been employed; the non-operative, and the operative. Among the former may be mentioned the long continued use of splints, massage, and manipulation, the use of fibrolysin, ionic medication, x-ray treatment, and the internal administration of thyroid extract. These may all be dismissed as of no value.

Dupuytren advocated an open transverse incision and allowed the wound to heal by granulation. This method would not be employed by modern surgeons.

William Adams practised subcutaneous division of the fascia followed by long continued use of splints. This method is open to the objections that it frequently fails entirely to relieve the condition, that relapses occur, that it occupies a long period of time during which the patient is unable to work, and that permanent stiffening of the fingers not infrequently results from the long splinting.

Paul Berger employs a wide excision of the skin and fascia and a transplantation of a skin flap from the chest. Professor J. Hutchinson says that this method has nothing to recommend it.

The remaining methods of operation have consisted in excision of the fascia by means of various flaps or longitudinal incisions. Kenneth Black says that "skin flaps are dissected from the palmar fascia, the fascia is excised, and the wound is closed. Greatest care in asepsis must be observed, for if the wound suppurates the operation will fail and the resulting contracture will be excessive. The operation is not easy, it requires patience. Dissection of the skin from the fascia is troublesome, and the excision of the fascia from the tendon sheaths must be done bit by bit. Then splints must be employed for weeks."

Professor J. Hutchinson (Hunterian Lecture, *Lancet*, 1917, i, 285) in one of the most recent articles on this subject states that: "(1) The main incision should be linear and vertical over and closely parallel to the ridge of palmar fascia. Small transverse incision at the front of the web may be needed to excise any prolongation of fascia in front of the first phalanx. (2) In the case of the middle and ring finger it is impossible to avoid placing the incision toward the mid-palm. In the case of the little finger alone the incision may be made rather to the ulnar side of the hand where the skin is more supple and less subject to pressure." He adds that V- and Y-incisions are not good as they cause too much scarring of the palm.

The advantage of an incision along a transverse crease of the palm is too evident and too well known to surgeons who have operated upon the hand to require discussion. The same principle applies to surgery in any portion of the body; an incision along a natural crease of the face, or the neck, or the elbow, or the wrist will occasion much less scar, keloid overgrowth, or contracture than an incision transverse to the creases. But I do not find in a search of the literature that any writer has advocated the use of such an incision for the excision of the palmar fascia. That the operation through such an incision is not unusually difficult is demonstrated in the case presented before you this evening.

In regard to the third step of the operation, the implantation of a free fat transplant, I was not aware at the time of operating upon this case that this procedure had been employed in Dupuytren's contracture. But I have since read an article by Dr. Alfred Peiser in *Zentralblat f. Chir.*, Leipzic, 1917, xliv, 6–8, in which he advocates the use of a free fat transplant following the excision of the fascia through a flap incision whose base is toward the thumb. He states that a search of the literature shows that the free fat transplant had not yet been used in Dupuytren's contracture.

Spitzy employed free fat transplants in cases of contracture of the fingers and hand other than Dupuytren's contracture (*Zeitschr. f. orth. Chir.*, Stuttg., 1915, xxxv, 550–561).

While I have operated upon but one case, and it of recent standing, by the method above described, and am familiar with the objections which may be offered on this account, nevertheless, I am convinced that this procedure is in accord with sound surgical principles which have been frequently demonstrated in my own experience and in that of many others in plastic surgery of the hand and forearm.

A CASE OF PARALYTIC CALCANEOCAVUS WITH EXTREME DEFORMITY TREATED BY JONES'S METHOD OF TARSECTOMY IN TWO STAGES[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

In most cases of calcaneocavus deformity following infantile paralysis, I adopt the operation of transverse horizontal section of the tarsus, as described by G. G. Davis. This operation combines a subastragalar arthrodesis with posterior displacement of the calcaneum and plantar half of the anterior tarsus, so that not only is the tendency to valgus remedied, but the leg bones are shifted forward to a position over the apex of the cavus deformity, with the result that weight-bearing keeps the sole flat. In the following case, however, the deformity was so extreme that I resolved to employ the operation described a number of years ago by Mr. Robert Jones, and which consists essentially in removing, as the first stage of the operation, a wedge of bone from the front of the tarsus, thus bringing the anterior tarsus and metatarsus into line with the calcaneum; while the second stage of operation comprises the removal of a similar wedge of bone from the posterior portion of the ankle, bringing the foot again into a normal position in relation to the leg.

Anna L. was seventeen years old when she came under my care at the Orthopædic Hospital in February, 1916. When two years of age she had had an attack of infantile paralysis, leaving her with a helplessly powerless left lower extremity. She had had no treatment for this condition until about a year before she came under my observation, when she had applied at the dispensary of a general hospital. Here an apparatus had been ordered for her, extending to the hip, and enabling her to walk without crutches, with her knee stiff.

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[1] Reprinted from the American Journal of Orthopedic Surgery, August, 1917, No. 8, xv, 602–607.

Examination disclosed a much shortened and atrophied left lower extremity, with fair power at the hip and in the hamstring muscles, but with no power in the quadriceps nor in any muscle below the knee except very slight power of extension and flexion of the toes, and in the tibialis anticus and peronei. There was an extreme degree of calcaneus and cavus deformity (Figs. 92 and 93). A skiagraph (Fig. 96) showed the calcaneum at an angle of 60 degrees with the anterior tarsus and the metatarsus.

Operation (March 1, 1916) was done at the Episcopal Hospital. An Esmarch band was applied above the knee. An incision 7.5 cm. long was made across the dorsum of the foot from the lateral margin of the extensor tendons to the lateral margin of the sole. The short extensors were detached from the bone and, together with the long extensors, were held aside by a retractor. A wedge of bone (about 60 degrees) was cut from the tarsus, the posterior section being at the midtarsal joint, and including the articular surfaces of the astragalus and calcaneum; while the anterior section passed through the cuneiforms (Fig. 96). By strongly flexing the foot it was brought into line with the calcaneum (Fig. 97). Tenotomy of the plantar fascia then made the sole almost flat (Fig. 95). The Esmarch band was removed before closing the soft parts with chromic gut sutures. The foot was dressed in plaster of Paris in extreme dorsiflexion (Fig. 94).

The gypsum case was removed seven weeks after operation and the wound found solidly healed.

The *second operation* was done April 26, 1916, about eight weeks after the first. Under Esmarch anemia, and with the patient prone on her face, a transverse elliptical incision was made over the back of the ankle region, excising an ellipse of skin 3 cm. wide and 10 cm. long. The very atrophic tendo Achillis was divided and its ends turned aside. The lower end of the tibia, the posterior surfaces of the astragalus and calcaneum were bared by displacing the flexor longus hallucis toward the median side of the ankle. A wedge of bone was cut from the posterior surface of the ankle, including most of the posterior part of the astragalus; the upper section included the posterior lip of the tibia, and the lower section shaved

Fig. 92

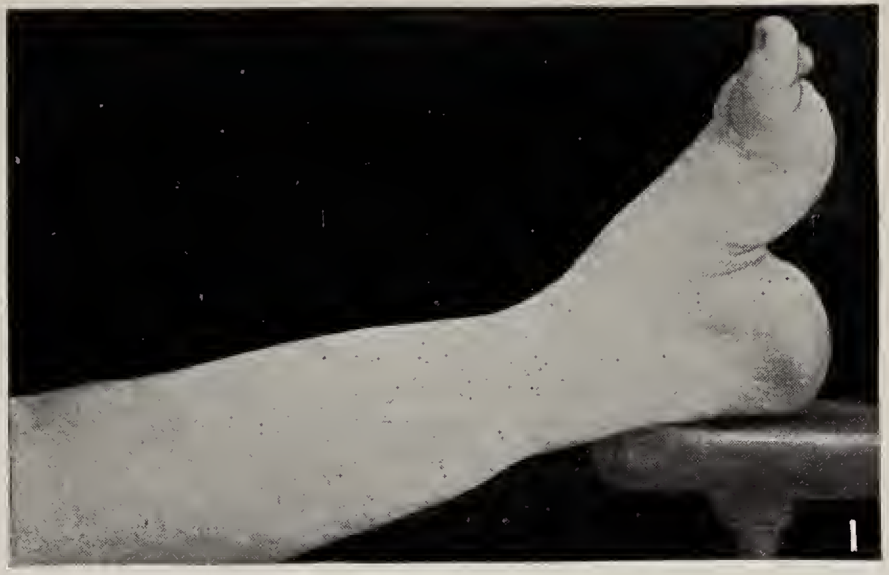
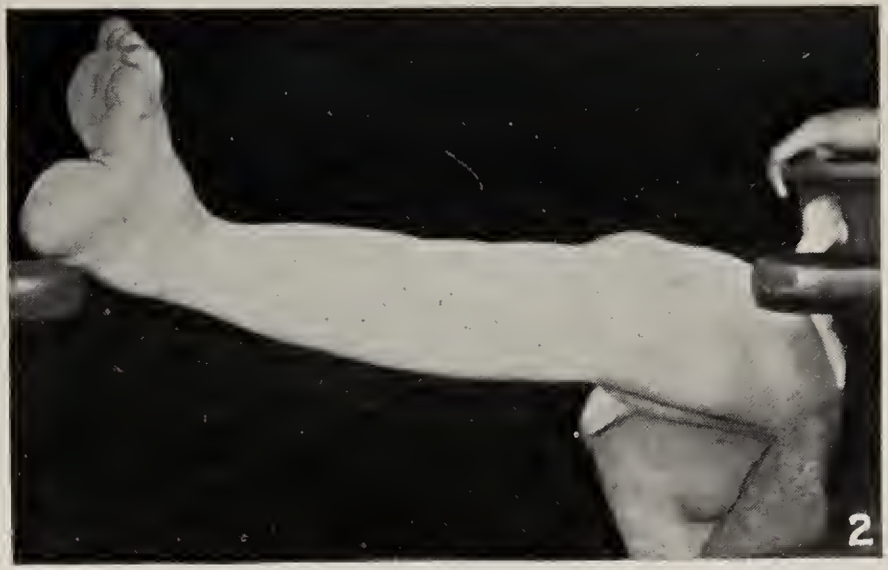


Fig. 93



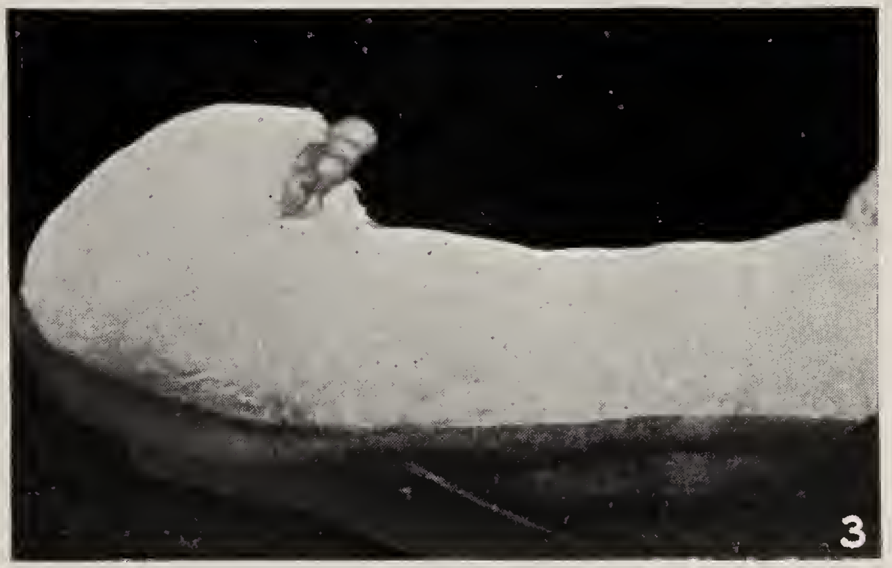


Fig. 94

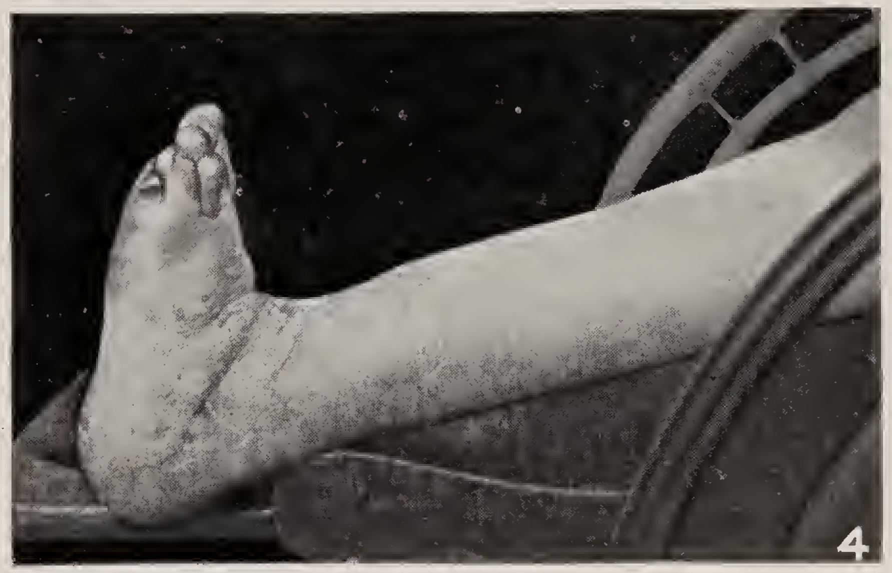


Fig. 95

Figs. 92, 93, 94, 95.—Case of calcaneocavus deformity in a girl, aged 17 years, following an attack of poliomyelitis at the age of 2 years. Had received no treatment. 1 and 2, before operation; 3, in gypsum case after first stage of operation; 4, result of first stage of operation.

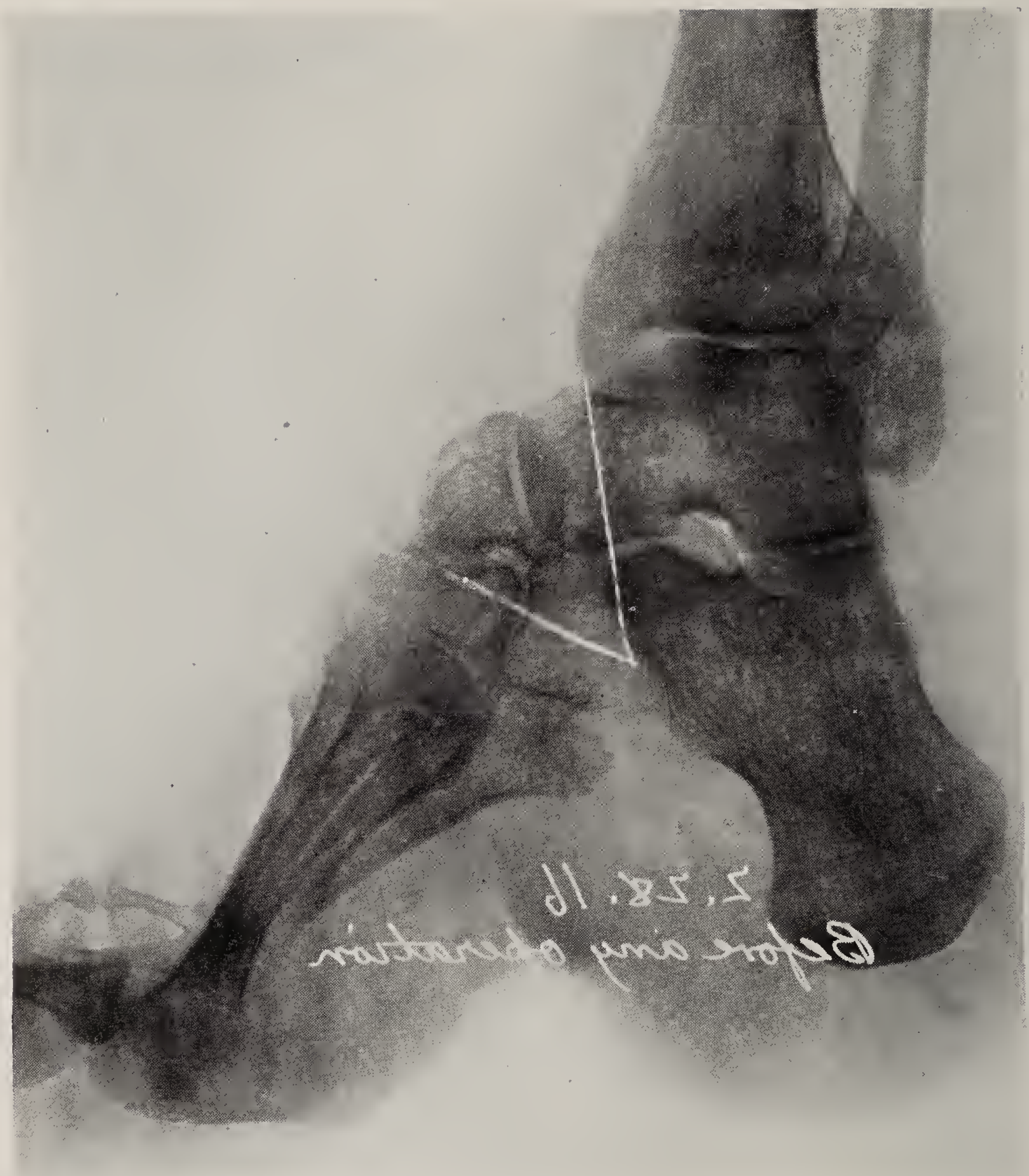


Fig. 96.—Skiagraph of foot showing extreme deformity (calcaneocavus). The proposed resection of the anterior tarsus is outlined in white.

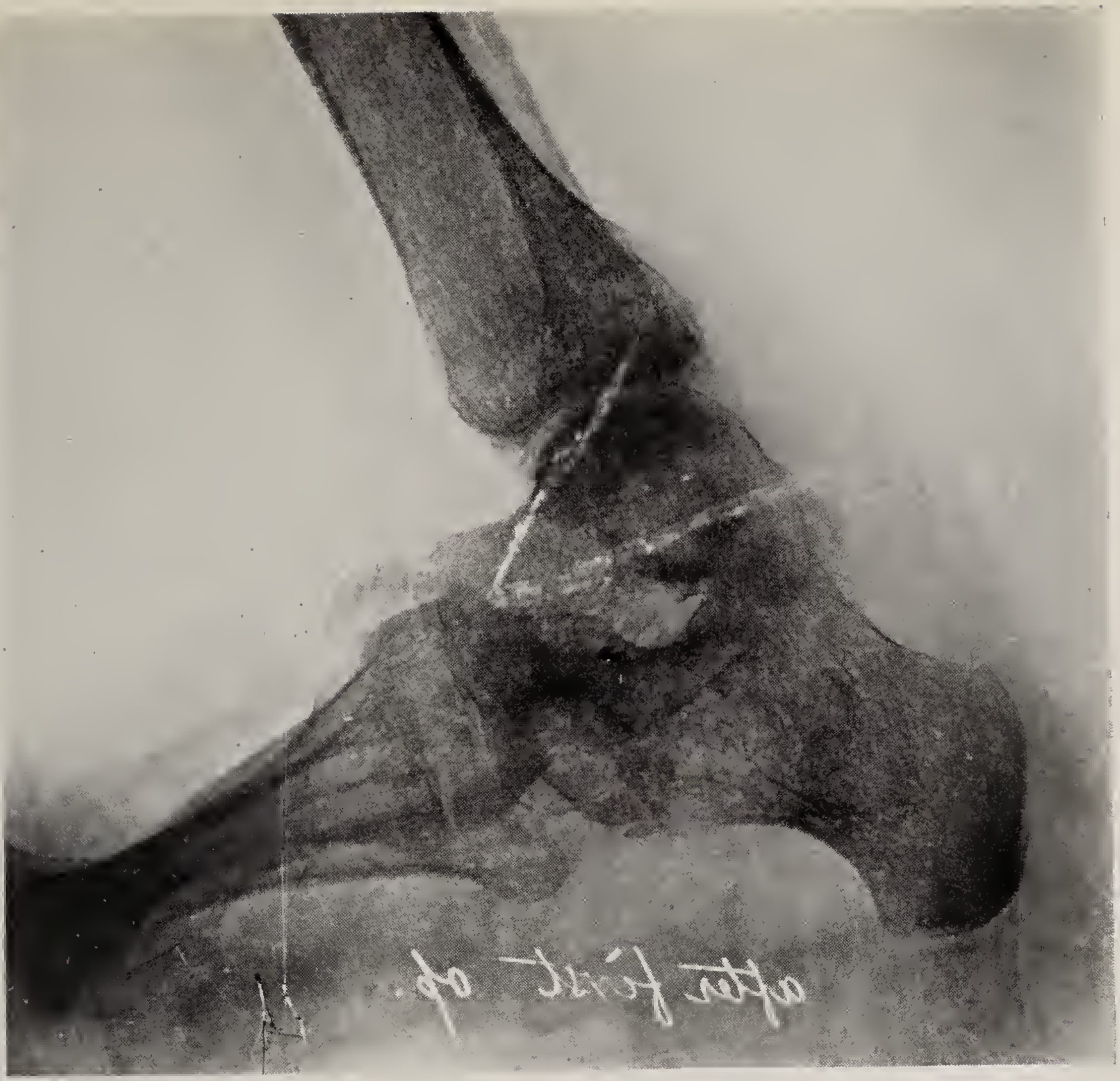


FIG. 97.—After first stage of operation. Cavus has been corrected. Proposed resection of posterior tarsus outlined in white.

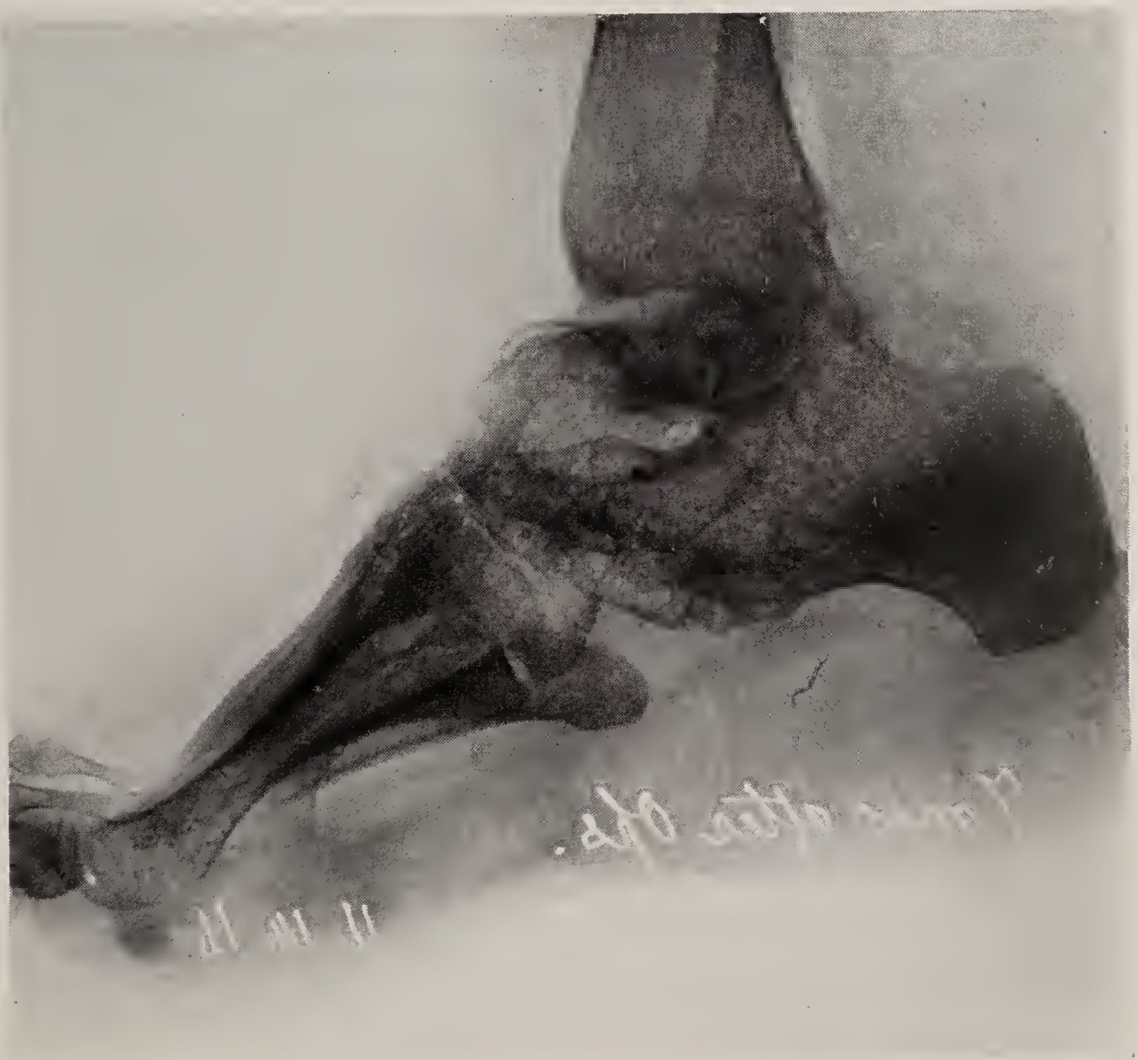


FIG. 98.—Seven months after second stage of operation. The foot is ankylosed at the ankle in a position of slight equinus, thus preventing collapse of the knee in walking. She walks well (without a brace).

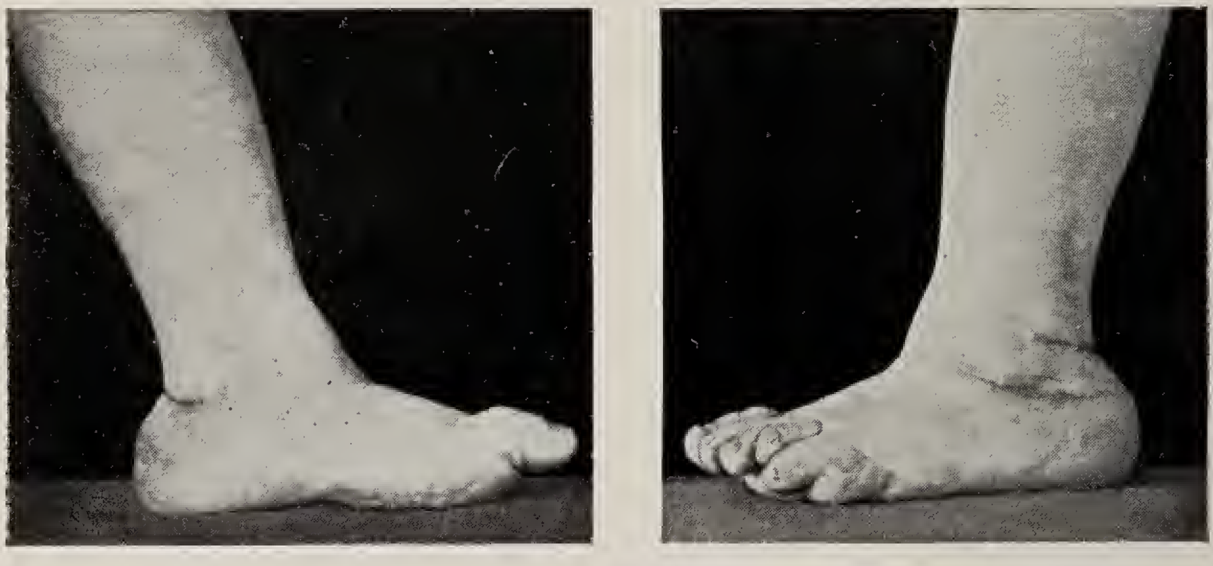


Fig. 99.—Seven months after operation. Walks without apparatus and has no pain at all, now nearly five years later.

off the upper surface of the calcaneum (Figs. 97 and 98). The plan was to make an arthrodesis of the ankle and subastragalar joints, with the foot in a position of slight equinus, in an effort to compensate in this way for the paralysis of the quadriceps femoris. The foot was thus brought up into a position of moderate equinus. Four or five bone chips, denuded of cartilage, were replaced to fill dead spaces. The tendo Achillis was sutured by overlapping its end, after two mattress sutures of No. 2 chromic gut had been passed to unite the calcaneum with the tibia. There was no bleeding on removal of the Esmarch, and the soft parts were closed with chromic gut sutures. The foot was dressed in a position of moderate equinus in plaster of Paris.

The first dressing was done about ten weeks later, when the cast was removed. The wound was healed. The old brace was temporarily reapplied in July, with the knee-joint locked and no joint at the ankle. This apparatus was worn until November, when weight-bearing without support was begun. In March she was able to walk well without any support; the foot was stable and entirely painless, and the position of slight equinus overcame the tendency to collapse of the knee. There is still moderate limp, but otherwise no disability.

The patient was shown, by invitation, at one of the sessions of the Interurban Orthopædic Club, in Philadelphia, April 21, 1917.

CASE OF JACKSONIAN EPILEPSY CAUSED BY BRAIN TUMOR—SUCCESSFUL REMOVAL OF THE TUMOR[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

Dr. Ashhurst reported the case of a patient, John T., aged thirty-one years, on whom he had recently operated at the Episcopal Hospital. The man was originally admitted to the Hospital November 22, 1919, complaining of "headache and stomach trouble," and was sent to the medical ward, then in charge of Dr. M. H. Fussell, and was eventually transferred to the surgical service January 3, 1920, by Dr. John B. Carson, who had succeeded Dr. Fussell on duty in the medical service.

Dr. Ashhurst saw the patient first in consultation on January 2, 1920, and learned the essential facts of the history as follows: The patient's family history was negative. He was unmarried. He was born in Poland and came to the United States in 1912. He was a laborer and his general health always had been good. He smoked and drank very little and had had no serious injuries and no operations. He denied venereal disease.

About a year ago the patient had his first convulsion, losing consciousness. Since then he had had eight other attacks of varying severity at intervals of weeks or months. Three weeks before admission (*i. e.*, about November 1, 1919) his family physician, Dr. Jacob B. Feldman, made a spinal puncture for a Wassermann test.[2] After this the patient developed headache and vomiting. In the three weeks elapsing between this visit to his physician and his admission to the hospital he had five convulsions. If he tried to leave his bed he vomited. After his admission to the hospital, during the latter part of November and during December, he had five other convulsions, one of which at least was determined to be Jacksonion in type, beginning in the left hand.

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[1] Presented before a joint meeting at Philadelphia of the Philadelphia Academy of Surgery, and the New York Surgical Society, February 2, 1920. Reprinted from Annals of Surgery, 1920, ii, 402.

[2] This was reported negative, as was the blood Wassermann after his admission to the hospital.

The patient, while on the medical service, was seen with Dr. Carson by Dr. Charles W. Burr, consulting neurologist to the hospital, who was unable to make a diagnosis. It was learned after operation that the patient's physician, Dr. Feldman, had called Dr. Alfred Gordon in consultation, and that Dr. Gordon had suggested an operation, on account of a bony lump in the skull in the right parietal region, and after seeing a skiagraph of the patient's head.

Examination, January 2, 1920, showed a young man, with rather exprēssionless face, who felt fairly well when lying quiet in bed, except for a constant terribly severe headache, in the frontal region and on the top of the head. The right eye was kept closed or nearly so, but could be opened. The pupils were equal and reacted (rather sluggishly) to light and accommodation. An examination of the eye-grounds made by Dr. Harold G. Goldberg, ophthalmologist to the hospital, on November 29, 1919, had shown that the right eye was negative but that the left eye showed slight paleness of the nerve and disturbance of retinal pigment, and one minute hemorrhage of considerable duration down to the temporal side of the disc. Dr. Goldberg considered it a beginning neuroretinitis. A subsequent examination by Dr. Goldberg, January 5, 1920, confirmed the above findings, but showed no changes since the previous examination.

There was no paralysis of any of the cranial nerves, and the chest, abdomen and genitalia were negative. The extremities also were negative except for slight accentuation of the knee-jerks. There was no Babinski, no ankle or patellar clonus, and no Kernig's sign.

In the right parietal region of the skull was a sessile exostosis, about 7.5 cm. in diameter, and raised about 5 mm. above the surrounding surface. Pressure on this lump caused some pain. He said he noticed the lump first about five years previously, and that it had been very slowly growing larger. Questioned as to any injury to this region, he said that when he first came to this country he attended night school, and there were many fights among those standing in line waiting for admission, but he did not remember any specific injury to his head. Skiagraphs made by Dr. R. S. Bromer, radiologist to the hospital, showed an area of rarefaction of the inner table of the skull corresponding to the site of the exostosis.

Here was a man, Dr. Ashhurst said, who besides having epilepsy, apparently Jacksonian in type, had intense and persistent headache, had unprovoked attacks of vomiting, had an abnormal bony lump over the right motor region, and who had changes in the left eyeground suggestive of increased intracranial tension. He considered these facts sufficient to justify an exploratory operation. His opinion was that the exostosis itself might be sufficient explanation for the symptoms.

Operation, January 6, 1920, ether (intrapharyngeal). Operator, Dr. Ashhurst, assistants, Dr. Mendel and Dr. McGuire. A bone flap three of whose sides measured about 8 cm. each, and whose base (in the temporal region) measured 3 cm. was turned down from the right motor region, the exostosis being about in the center of the flap. Bleeding from the scalp was controlled by Kocher hemostats. The bone flap was cut by means of the Hudson trephine, Gigli saw and De Vilbiss forceps. After raising the bone flap its under surface over the area corresponding to the exostosis was found eroded and hemorrhagic in appearance. The bone flap was therefore removed, together with a portion of the cranial aponeurosis, which was adherent to the underlying exostosis (Plate I). (Time elapsed at this stage of the operation fifty minutes.) The exposed dura was granular over an area corresponding to the exostosis, and it was determined to excise it. It felt a little less resistant than the surrounding exposed dura, but the entire surface exposed pulsated normally. After ligation of four or five meningeal vessels, with fine silk on a curved needle, the dura was opened, a grooved director slipped inside and a rectangular incision (5 by 5½ cm.) was made with a scalpel, upon the director, entirely surrounding the diseased dura. As an attempt was then made to raise the diseased dura by mouse-toothed dissecting forceps, it was found to be continuous with a tumor embedded in the brain. By careful preliminary double ligation (with fine silk or a curved needle) of all pial vessels entering the tumor, and dividing the vessels between the double ligatures, it was possible very slowly and cautiously to outline the tumor. By making gentle traction on the edges of the dura, and wiping away the convolutions of the brain with wisps of cotton (moistened in extremely hot salt solution) held in the fingers, the brain tissue very gradually was peeled off the tumor, millimeter by millimeter, the union between the two being like that of inflammatory lymph adherent to the intestines, and the friability of both brain and tumor being nearly as great as that of the flakes

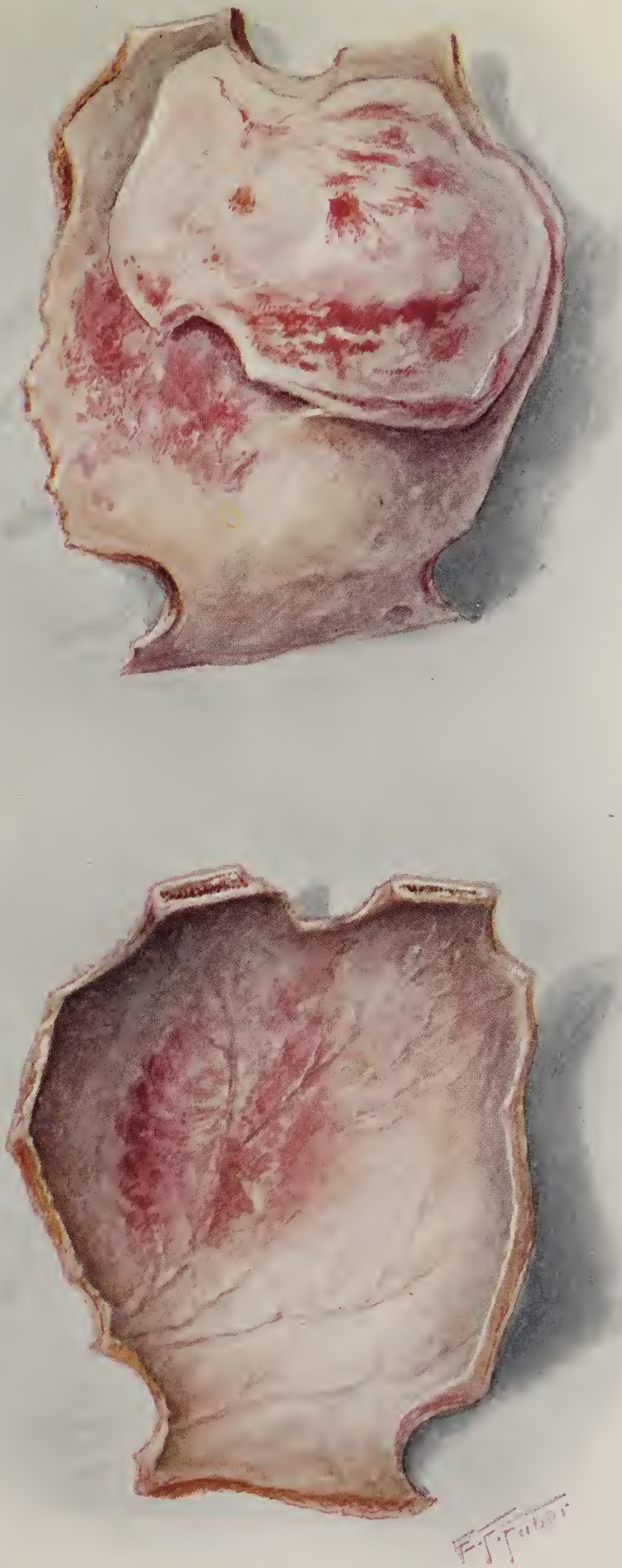

Plate I.—Portion of cranium forming osteoplastic flap excised, showing roughening on its inner surface overlying the brain tumor.

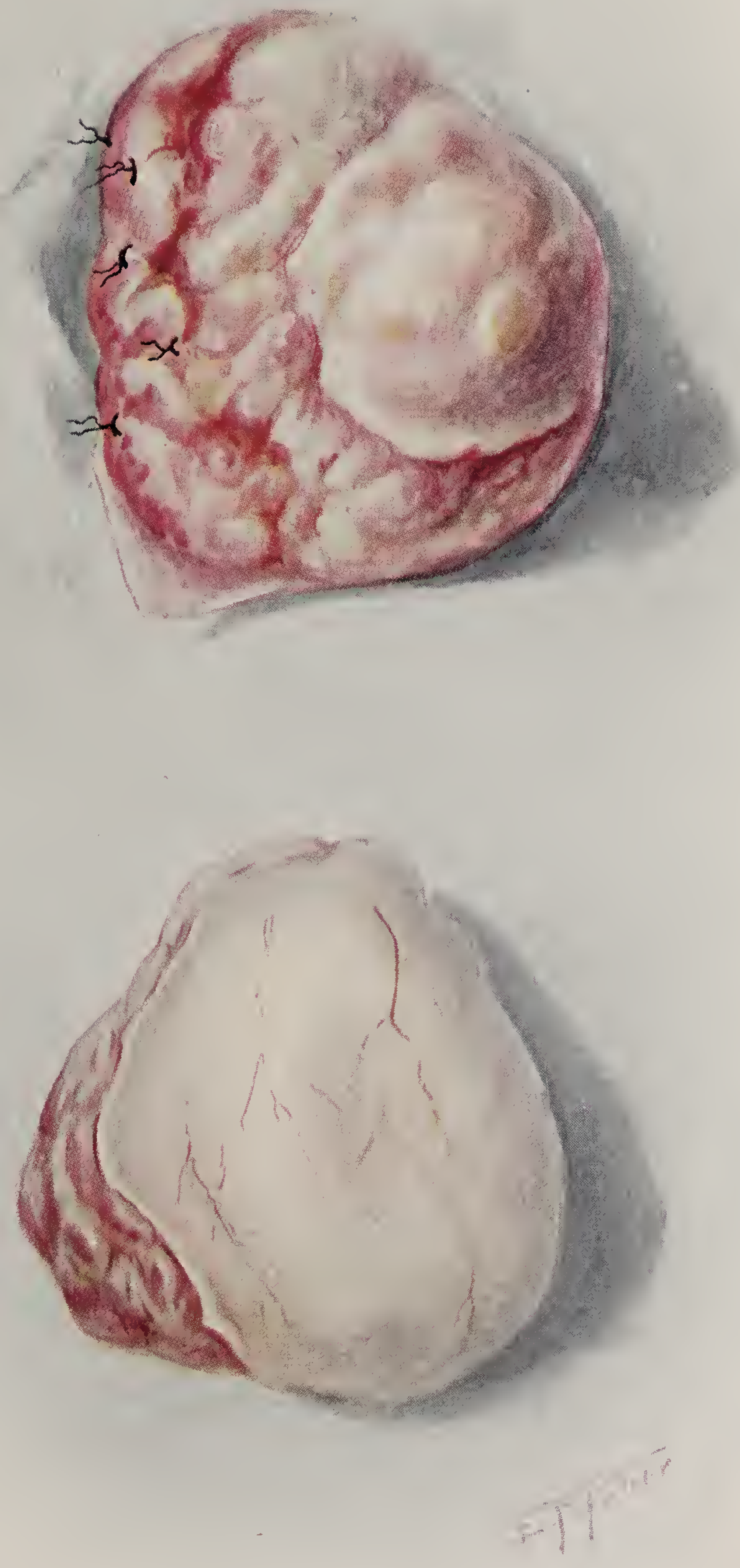

PLATE II.—Endothelioma removed from right hemisphere of cerebrum Cerebral surface is shown above; the subcranial surface, with the attached dura, is shown below.

of inflammatory lymph. Only at one point was any apparent damage done to the brain, when the needle passed around one of the pial vessels pricked the cortex, which immediately became suffused with a pink color over an area about 1 cm. in diameter. After the region of pial circulation was passed, the remainder of the stage of enucleation of the tumor was almost bloodless, any very minute ooze being promptly controlled by application of wisps of cotton squeezed out of an almost boiling saline solution. The tumor measured 5.5 cm. by 5 cm. on its surface, and was 4 cm. deep[1]. Plate II. The hollow left in the brain pulsated normally, but showed no immediate tendency to fill up (Fig. 100). (Time elapsed at this stage of the operation over two hours, about an hour being consumed in enucleating the tumor from the brain.)

During the removal of the tumor, Dr. McGuire cut a free transplant of fascia lata from the patient's right thigh, and this transplant (a rectangle 5 by 5.5 cm.) was sutured to the edges of the defect in the dura by interrupted silk sutures, and was gently pressed down into the hollow left in the brain. The flap of scalp was then replaced and accurately sutured (without drainage) by interrupted sutures of silkworm gut, the sutures being placed about 0.5 cm. apart, and thus effectually controlling all bleeding from the scalp.

The patient's condition was good throughout the operation, which was three hours in duration, and he left the table with a pulse of 128. At no time subsequently were there any unfavorable symptoms. For the first twenty-four hours after operation he said his left forearm and hand felt numb, but at the end of this time normal sensation returned to all but thumb and index finger. By January 18, 1920 (twelve days after operation), the index finger felt nearly normal, but the thumb still felt anesthetic, though both index finger and thumb distinguished pin contact throughout. At this time the patient was fairly convalescent. He seemed much brighter than before operation, had no headache at all, but still felt giddy on sudden movements or on attempts to sit up in bed.

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[1] The specimen was submitted to Dr. William G. Spiller, at the University of Pennsylvania, who reported, after examining it microscopically, that it could be only one of two things—an endothelioma or a fibroma; and he was inclined to class it as the former. He was further of the opinion that such growths in the brain were frequently the result of irritation from a lesion of the skull, originally traumatic in origin. This had been Dr. Ashhurst's own belief at the time of operation, and he was glad to have it confirmed by so able an authority as Prof. Spiller. Krause (Surgery of the Brain and Spinal Cord, translated by H. A. Haubold; New York, 1909, vol. i, p. 71), however, held the opposing view, that tumors of the dura were responsible for the growth of new bone in the skull. The latter view would justify one in replacing the bone flap after removal of the tumor; whereas if the former is the correct theory, replacement of the same bone might in time cause another tumor to form.

In 1899, Spiller reported a case similar to the one now recorded, and in 1907 reported a second case and reviewed the literature (Jour. Am. Med. Assn., 1907, ii, 2059). In advanced cases the thickening of the skull overlying the tumor becomes so widespread as to justify the name of *hemicraniosis* applied to it in 1903 by Brissaud and Lereboullet.

January 28. Sitting up in chair.

February 2. Four weeks after operation, walking about ward. Has had no convulsions since operation. If symptoms develop later from the skull defect caused by removal of the bone flap, it is planned to do a second operation and fill the opening by a bone transplant from the outer surface of the neighboring skull.

June 7. Five months since operation. Has gained thirty pounds in weight. Has had no convulsions since the operation. His ears no longer buzz, but he has some consciousness of his brain in stooping (he has returned to his work as automobile mechanic) and in walking fast. On June 10 a bone transplant was cut from the left parietal region, consisting of the outer layer of the skull and overlying pericranium, and was inserted into the defect in the right parietal region which had been previously prepared for its reception. The patient, who was receiving ether by intrapharyngeal insufflation, in the head high position, was in good condition until the head was turned over to the left, for insertion of the transplant in the right. His condition at once became unsatisfactory, and in spite of stimulation he died, apparently of respiratory failure, as the scalp was being sutured. The duration of the operation was nearly two hours, but there was no evidence of shock: he ceased breathing some seconds before the heart beat stopped, and artificial respiration and cardiac massage (through the unopened chest and abdomen) were of no avail. No autopsy was permitted.

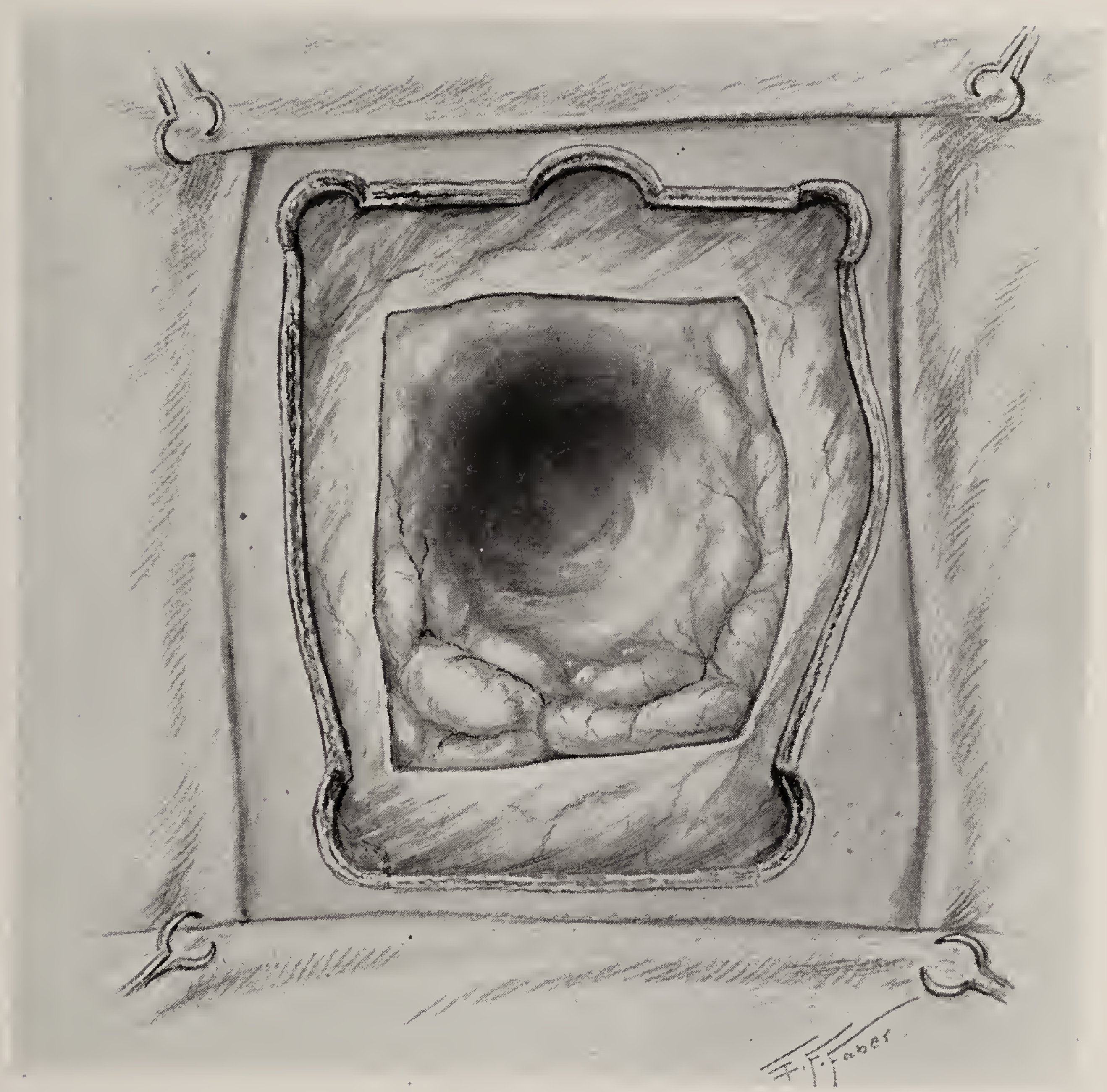

FIG. 100.—Cavity left on surface of brain after removal of tumor. (After a sketch by Dr. James H. Mendell.)

A CASE OF HEMATOMYELIA, WITH CROSSED PARALYSIS (SENSATION ON LEFT, MOTION ON RIGHT)[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

Dr. Ashhurst also presented notes of the following case. He found the patient in the ward at the Episcopal Hospital when he took over Dr. Frazier's service from Dr. Mutschler, August 1, 1914, and was indebted to these gentlemen for the privilege of reporting the case. For the ward notes he was indebted to Drs. J. Walker Moore and J. P. Jones, the surgical interns.

The patient, a young man, aged twenty-five years, was admitted July 23, 1914, and discharged October 29, 1914. On the day of admission, at 7.30 A.M., he had been knocked backward from the deck of a ship to the deck beneath, landing on his forehead. He was not unconscious at all. He had no pain for the first five minutes, but then felt intense pain all over the body. After the fall he was unable to move a muscle of his body below the chin, but he could talk and move his eyes and eyelids normally.

On admission, at 2 P.M. the patient is paralyzed below the sixth cervical vertebra. He has great difficulty in breathing. He can talk, open his mouth and protrude his tongue, but he is unable to move his head or the rest of his body. There is no bleeding from the ear or nose.

He was placed on a water bed.

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[1] Presented at a joint meeting of the Philadelphia Academy of Surgery and the New York Surgical Society, held at Philadelphia, February 2, 1920. Reprinted from Annals of Surgery, 1920, ii, 406.

Physical Examination. Fairly well nourished and developed young fellow. Temperature, 102° to 101° F.

Head: Eyes react normally to light and accommodation; pupils are equal. Nose and ears are negative. Tongue is moist and clean; no paralysis. There is an abrasion and some slight swelling of the forehead. Complains of intense pain in the neck when the head is moved. There is no displacement of the spinous processes of the cervical vertebræ. He is unable to move his head at all.

Chest: The breathing is shallow and difficult. Lungs and heart are normal.

Abdomen: No respiratory movements of the abdominal muscles. There is a scar of a hernia operation in the right inguinal region.

Extremities: All four extremities are paralyzed. The patellar and cremasteric reflexes are absent. The hamstrings and calf muscles contract when the overlying skin is pricked with a pin. The toes move when the sole of the foot is touched. There is loss of sense of pin-prick on the entire left side up to the neck, but this sense is retained on the right side.

Six hours later: Slight motion is perceptible in the left arm and the left leg; sensation remains about the same.

Ten hours after admission: Is able to move the right arm also; can flex but not extend the elbow. Unable to move the right leg. Breathing is better. Pain is less. Knee-jerks still absent.

7.24. Can move the right arm slightly. Has to be catheterized. No other changes. Temperature, 101° to 100°.

7.25. Given a purge.

7.26. Voided urine and has control of his bowels. Slight increase in power of triceps in left arm. Fibrillary contraction of both lower extremities. Still unable to move right leg. Cannot move the muscles of shoulder girdle.

7.28. Can move his shoulder muscles; no other change.

7.30. Temperature has reached normal.

8.1. Notes by Dr. Ashhurst.

Can rotate, flex and extend the head, but not strongly.

Right upper extremity: Can move the right shoulder. Trapezius, deltoid and axillary fold muscles all act slightly. Subscapularis good. Biceps good. Triceps no power. No power in wrist or fingers.

Left upper extremity: Shoulder muscles are all good. Elbow good but weak. Slight power in the extensors of the wrist and fingers.

Left lower extremity: Fair power throughout. Increased reflexes. Ankle clonus and Babinski present.

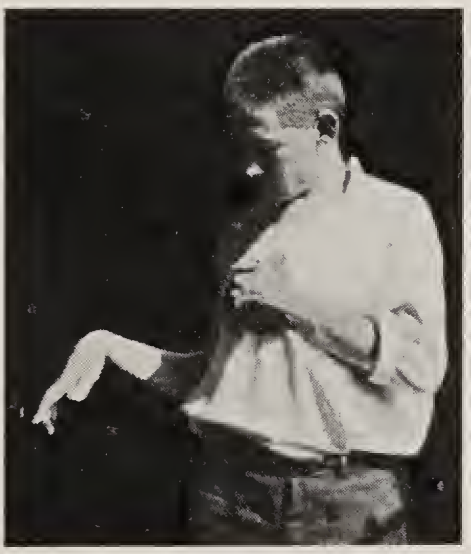

FIG. 101.—Residual paralysis following contusion of cervical portion of spinal cord. (hematomyelia.)

Right lower extremity: No power; spastic. Increased reflexes. Babinski present. Fibrillary contractures.

Sensation: Over the entire left side from the ninth intercostal space down, sharp and dull, are called dull. The remainder of the body has normal perception of sharp and dull. Over the left lower extremity cannot distinguish hot from cold. Over the left abdomen and thorax both hot and cold are felt as hot. Elsewhere sensation of heat and cold are normal.

Neck: Third cervical spinous process appears to be more prominent than normally.

Diagnosis: Hemorrhage into substance of cervical cord; hematomyelia.

8.3. Can flex right knee and hip with ease. No other changes.

8.4. Skiagraphs negative for fracture of cervical spine.

8.13. Can turn from side to side in bed and raise entire body from bed. Sensation continues impaired on left side. Is moved from water bed to ordinary mattress.

8.15. Good movements in arms and legs but very little power in the hands.

8.18. Can sit up in bed. Hands still very weak but arms are becoming stronger. Perfect movement in lower extremities.

8.21. Gradual improvement in all movements except the right hand.

8.29. Can flex right index finger but can move none of the other fingers or wrist on right. Can move all the right toes but cannot move the ankle-joint. The reflexes on right are greatly increased. The left extremities are normal.

9.2. Able to move right thumb.

9.10. Gradually getting more motion in fingers. Has now limited use of all fingers of the right hand. Walks all over the ward with spastic gait.

9.14. Except for delayed sensation in left leg and foot, sensation is normal all over body.

9.20. Goes up and down stairs, but with difficulty. Holds right hand flexed at wrist, with metacarpophalangeal joints extended (Fig. 101).

10.20. Very little spasticity in gait. Sensation now normal in left leg.

10.29. Discharged and referred to Orthopedic Dispensary.

July 9, 1917. This date (three years after injury) he paid his first visit to the Orthopedic Dispensary of the Episcopal Hospital, when the following notes were made by Dr. Gill.

Has some atrophy of essential muscles of right hand, with a tendency to contracture. Has weakness of triceps of right arm. Active abduction of right shoulder is slightly limited. Has loss of temperature sense on entire left side of body except left hand and forearm. Has loss of tactile sensation on right side. He is unable to raise toes of right foot from floor. Works as toolmaker.

Proper treatment was advised, but the patient never returned, and cannot now be found; though it is known he was a visitor to a patient in the ward less than a year ago.

IMPROVED METHOD FOR TOTAL ENUCLEATION OF EYEBALL[1]

By FREDERICK KRAUSS, M.D.
OPHTHALMIC SURGEON TO THE HOSPITAL

THE operation here described aims at preserving the relations of the recti tendons with Ténon's capsule.

The operation of simple enucleation of the eyeball has been always considered an easy one, and frequently assigned to the resident. The general surgeon has felt that it was within his field of action. The result is usually a permanent deformity to the individual thus operated. To overcome this deformity, the operation has been modified by implantations of foreign bodies in Ténon's capsule or sclera, in various ways. A very ingenious method is that recently suggested by Dimitry. (*Am. Jour. Oph.*, v. 2, p. 653.)

The modifications that I now present were suggested by the fact that in ordinary tenotomy, we will say, of the internal rectus, if a free incision is made in Ténon's capsule after the separation of the internal rectus from the sclera, the result will be almost complete abolition of the movement inward of that eye.

The same thing is done to all the eye muscles and Ténon's capsule in the ordinary method of enucleation. This can be overcome by using catgut sutures through the lateral muscle, attaching it to the Ténon's capsule of the opposite side, thus completely closing the space formerly occupied by the eyeball.

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[1] Reprinted from the Amer. Jour. Oph., March, 1920.

Secondly: Ténon's capsule can be carefully separated so that practically none of Ténon's capsule is sacrificed, and consequently there is no tension, but quick healing. To do this it is necessary that the conjunctiva be primarily dissected loose from Ténon's capsule into the fornix. After the wound in Ténon's capsule is closed, the conjunctival wound is closed with catgut sutures at right angles to the opening in Ténon's capsule.

This allows the conjunctiva to seek its correct adhesion on the mobile Ténon's capsule, which latter is filled with a blood clot which probably organizes, and forms at least a temporary filling of Ténon's space, replacing the fat, tendon, glass or gold ball of implantation operations.

The advantages are:

1. The full vitality and strength of the muscles of the eyeball are retained, giving movable socket with a minimum of retraction, or falling of the upper lid.

2. In addition to the excellent stump obtained, there is no violent reaction following this operation, as there often is in the gold ball implantation in the sclera.

3. There is no danger of extrusion or sympathetic ophthalmia, as there is at times in the implantation of a foreign body in Ténon's capsule.

4. There is no after bleeding or secretion, and a welcome absence of the catarrh that so frequently follows an enucleation.

The improvements that I suggest are in the method of application of the catgut sutures to the recti and Ténon's capsule—in the care to preserve entirely Ténon's capsule by separate dissection—finally covering the same with a separately detached conjunctiva.

I realize that it is difficult to suggest anything absolutely new in ophthalmic surgery, and others may have used this method of operating. The result is such a remarkable improvement that I beg to have you try it.

Operation. The patient is prepared in the routine way for ether operation. The local field is prepared to produce aseptic results according to custom.

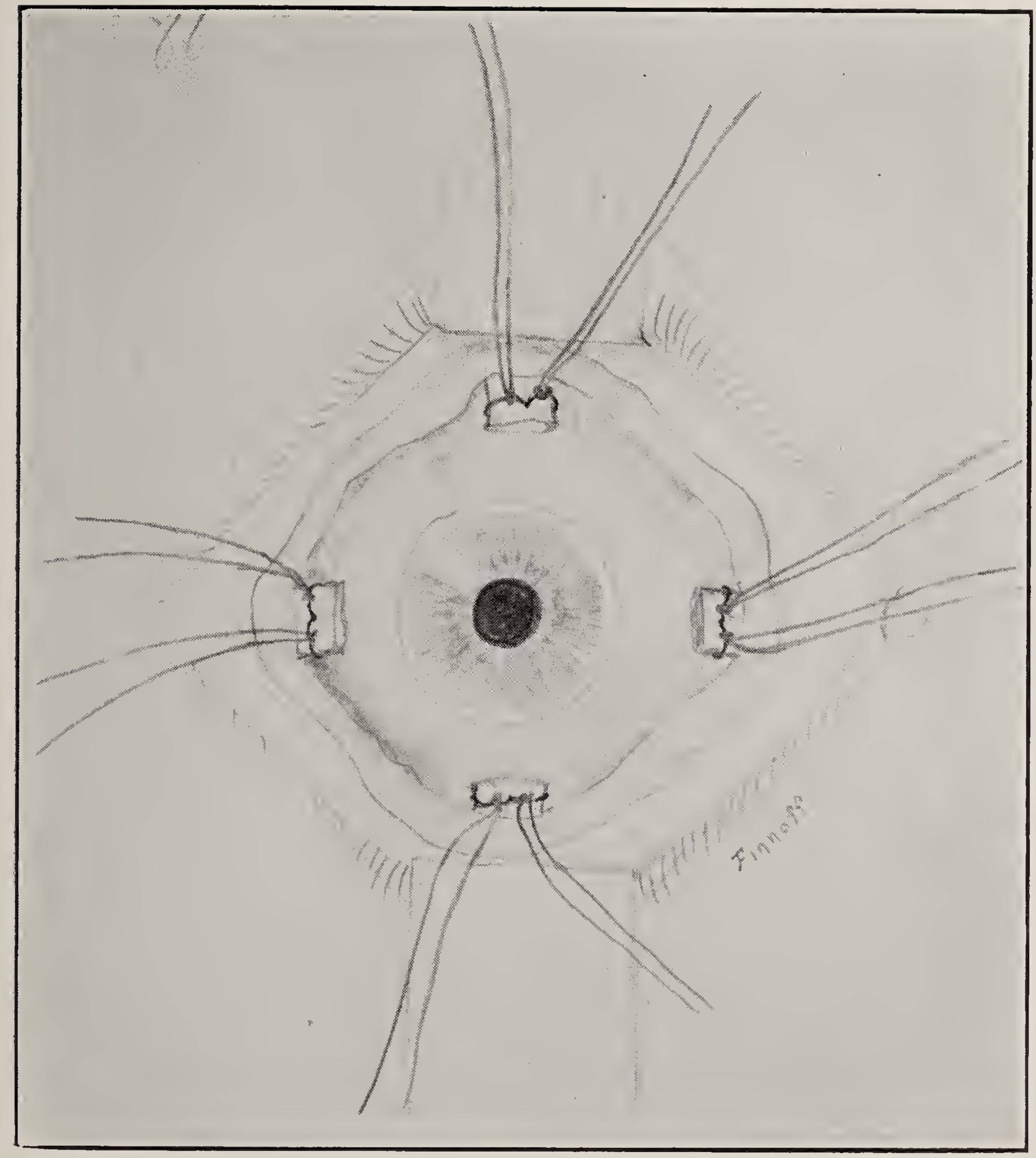

Fig. 102.—Conjunctiva dissected from Ténon's capsule. Sutures placed in recti tendons and tendons divided.

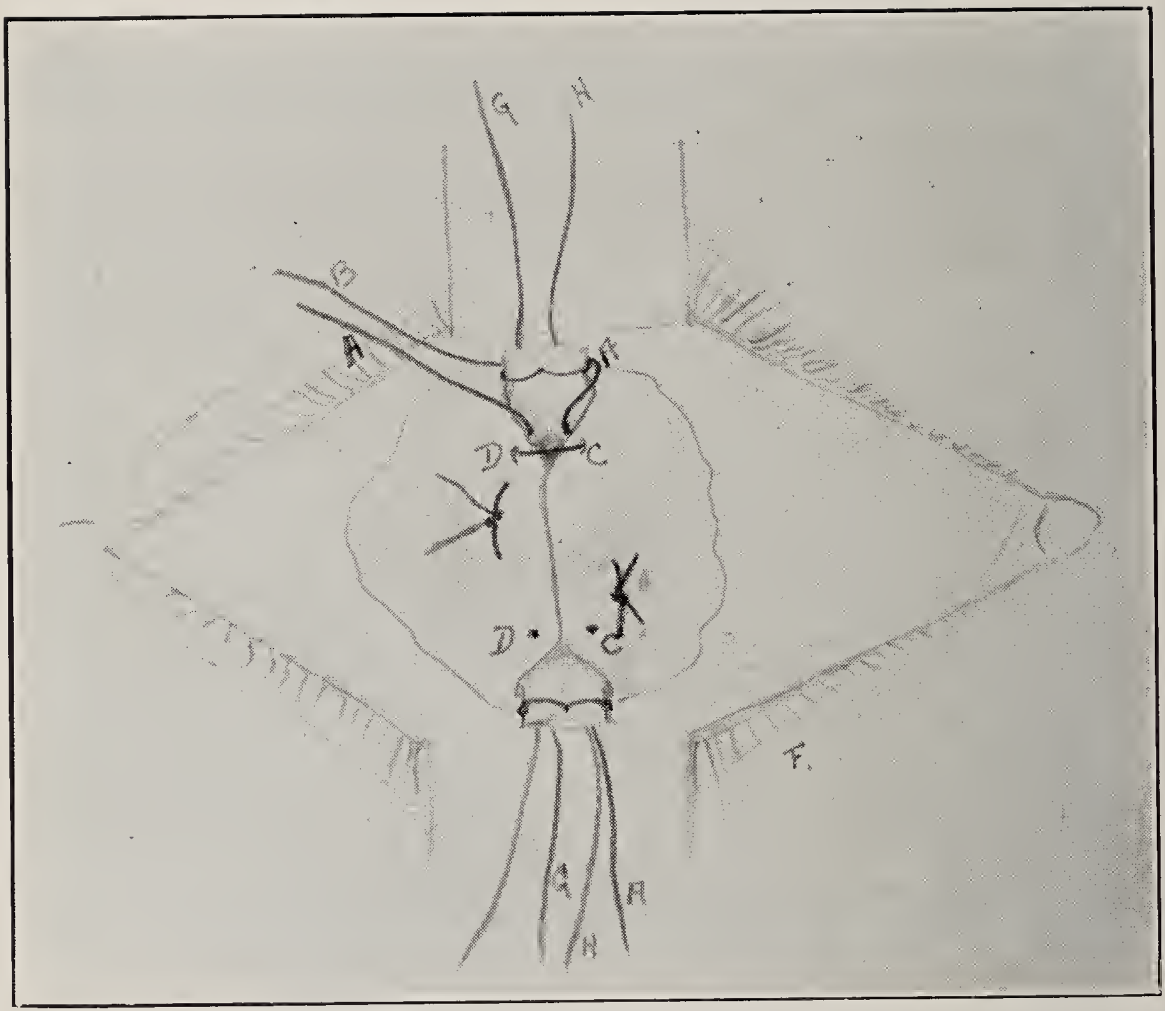

FIG. 103.—After enucleation, sutures from internus pass through Ténon's capsule to temporal side and tied; sutures of externus pass through Ténon's capsule to nasal side and tied. One thread from superior rectus starting at *A* is carried through capsule from within outward at *C*, then, from without inward at *D*, and finally tied to *B*, after *G* and *H* are cut short, burying the end of this tendon. The inferior rectus tendon is to be treated the same way, the suture *A* to be carried beneath the capsule brought out at *C*, introduced again at *D* and brought from beneath the capsule to be tied to *B*.

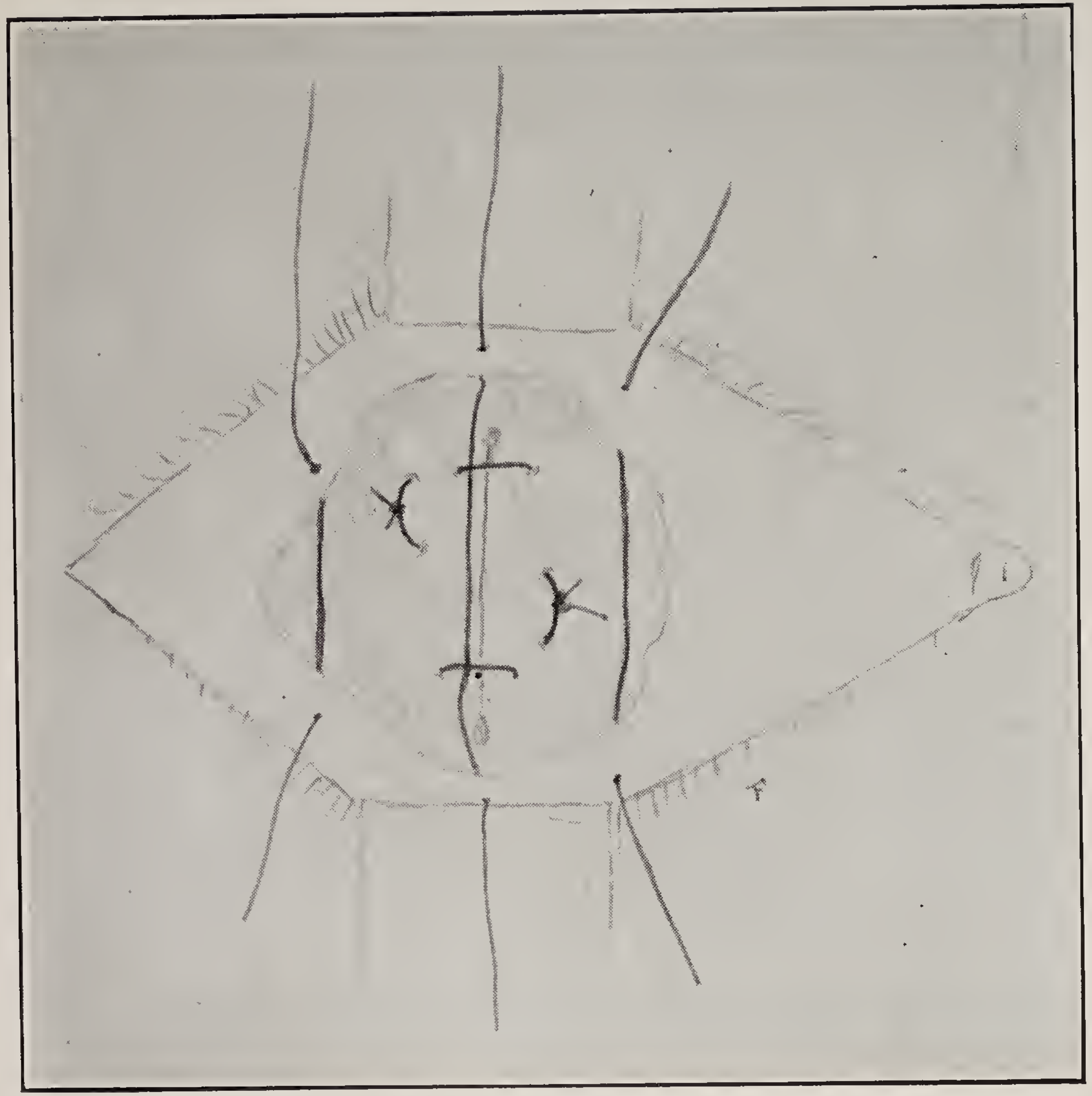

Fig. 104.—Shows sutures tied, closing the capsule, and others placed for closing the conjunctiva.

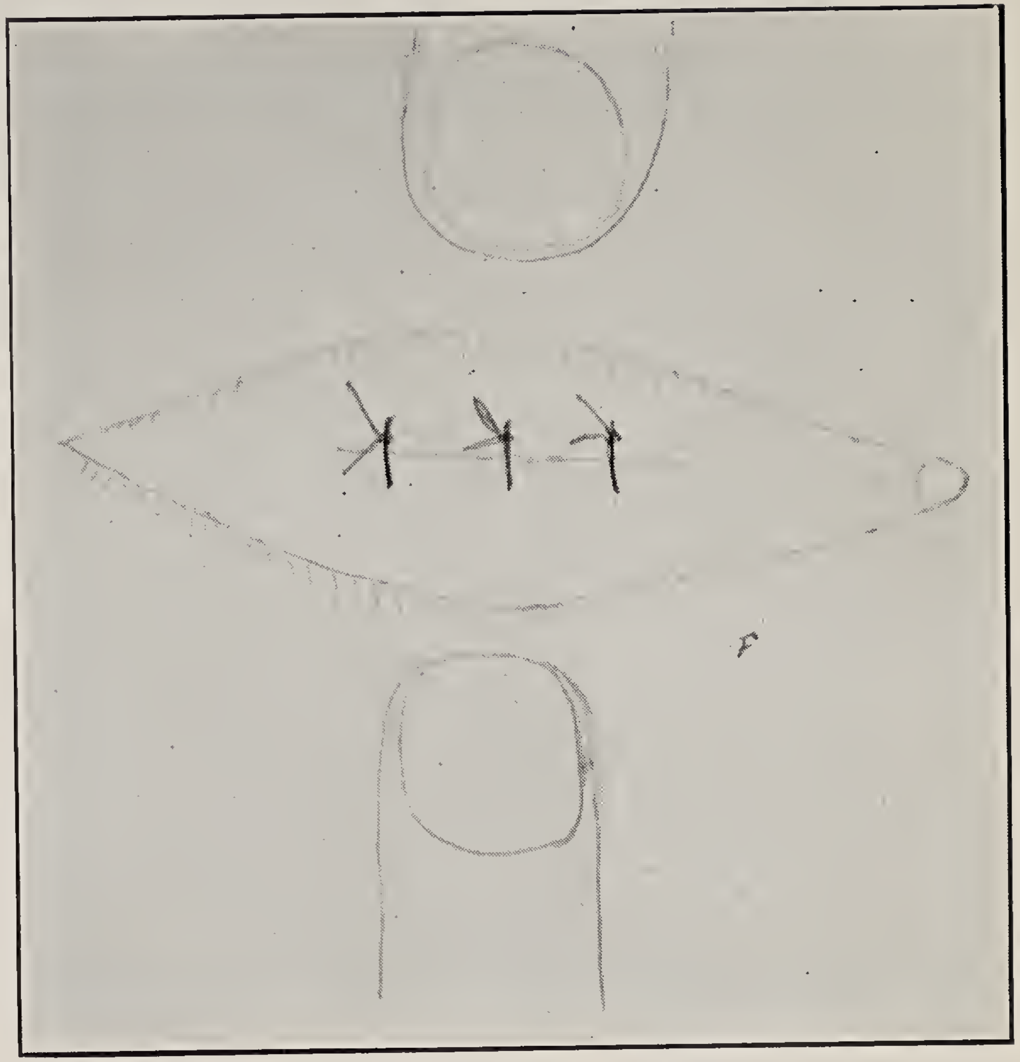

Fig. 105.—The conjunctival sutures tied, completing the operation.

After introduction of the eye speculum, the conjunctiva is carefully dissected from close to the cornea back into the fornix until it is entirely free from Ténon's capsule.

The internal rectus is caught upon a hook, Ténon's capsule slit parallel to the tendon close to its insertion, so that the hook is perfectly free in that position.

A catgut thread, No. 0 size (not chromicized) is threaded with three needles. The first needle is passed under the muscle to the opposite side, the second needle is passed through the center of the muscle. The loop thus formed is cut close to this second needle. The ends are then tied over the upper and lower half of the muscles respectively, the ends of the catgut being retained.

This preparation is similar to that usually taken in advancement operations of the muscles of the eye. The tendon is then cut close to the eyeball and the ends of the catgut caught by a hemostat, and placed out of the way. The recti muscles are all treated in the same way, giving the result shown in Fig. 102.

The capsule of Ténon is still intact if care has been taken to dissect only the conjunctiva as advised. The capsule is then carefully separated from the eyeball by introduction of a curved pair of scissors, beginning at the insertion of the internal rectus, hugging the eyeball. After complete separation, the eyeball can be enucleated in the usual manner by cutting of the optic nerve.

After primary bleeding has ceased, under pressure, the Ténon's capsule is filled with a blood clot, and is readily outlined by tension on the sutures introduced through the muscles. The wound is then sutured as follows: The catgut sutures of the internal rectus are passed through the Ténon's capsule of the opposite side directly below the insertion of the externus and tied. The sutures of the externus are then passed through the Ténon's capsule of the opposite side and tied, thus closing in the central part of Ténon's space containing the blood clot. The somewhat triangular spaces above and below these sutures are closed by the superior and inferior recti sutures, in the following manner: The one catgut holding the superior rectus tendon is passed through the neighboring capsule from within, out—then through the capsule on the opposite side of this triangular space, and tied to another thread still attached to the superior rectus.

This forms a sort of purse-string suture, completely closing the upper opening in Ténon's capsule. The same thing is done to close the lower opening. We then have a closed Ténon's capsule with the muscles in as nearly normal position as possible. The internus is given the preference in the attachment to Ténon's capsule as it is the strongest and has the most motion.

Finally the conjunctival wound is closed horizontally, burying Ténon's capsule. Both eyes are kept bandaged for forty-eight hours, as a precautionary measure.

The appearance of the socket after this operation presents a clean surface with no granulating areas and no bleeding. Healing is by first intention with perfect motility, due to the normal attachments of the muscles.

COCAIN POISONING IN OPHTHALMIC SURGERY

REPORT OF A CASE IN WHICH COCAIN POISONING TOOK PLACE AFTER THE INSTILLATION INTO THE EYE OF A FOUR PER CENT. SOLUTION IN PREPARATION FOR THE EXTRACTION OF A CATARACT

By HAROLD G. GOLDBERG, M.D.
OPHTHALMIC SURGEON TO THE HOSPITAL

The case to be described is a woman, aged sixty-eight years, white, a native of Scotland. The patient's husband is living and well; she has five sisters, all of whom are married and are well; seven sisters are dead from unknown causes. She has had one miscarriage. The family history for serious constitutional diseases is apparently negative. She states that she came to this country fourteen years ago. She does not recall ever having had the usual diseases of childhood, has always enjoyed excellent health, denies venereal infection and is not addicted to the use of narcotics or any other drugs, including alcohol. There is no history of any major operations or injuries. She was operated on a little over a month previously for preliminary iridectomy, which operation was entirely uneventful.

At the time of the patient's admission for the second operation, (cataract extraction) the vision in the left eye equalled light perception, and in the right eye, finger counting at a few feet, the first disturbance in vision being noted about six years before this date. The eyes in every other respect were negative, and the patient was considered a good risk for the cataract extraction. A general physical examination was likewise negative.

Operation. Local anesthesia of the eye to be operated upon was to be produced by the instillation of 4 per cent. cocain hydrochloride solution at two-minute intervals for three instillations. This is the usual method of producing anesthesia, but in this case five or six instillations were made because of some delay in the preparation of the instruments and dressings. Anesthesia having been produced the operation of extraction of the lens was proceeded with and carried to completion without accident of any kind. Immediately after the expulsion of the lens, however, and before the final toilet of the eye had been accomplished, the patient began screaming and attempted to leap from the table. She showed great excitement, expressed a desire to vomit, complained of excessive pain in the muscles of the back and neck and suddenly became extremely cyanotic, assuming a rigid opisthotonos position upon the table. Respirations and heart actions ceased, and the whole general aspect of the patient was most alarming. Morphin sulphate and camphorated oil were administered hypodermically, the muscles of the back and chest were massaged, and after several minutes the attack passed off and the dressing of the eye was completed, the patient apparently none the worse for her experience.

There are a number of interesting and instructive points in connection with this case. First of all it must be remembered that the patient had already had one operation performed under cocain anesthesia only a short time before the one just described. She was a perfectly well-behaved patient, not in the least nervous or apprehensive at the time of either operation. In the second operation the cocain instillations were continued over a longer period of time than usual, a greater number of drops were used, and lying upon the operating table in a supine position, no doubt some of the fluid passed through the tear duct into the nose and throat, where it was finally absorbed.

The second point of interest is the prompt recovery from the attack, especially without injury to the operated eye. During the attack the eye was supported as much as possible by keeping it firmly covered with a moist compress held tightly in place by the hand. It was a great surprise and the operator was very much relieved to find that the eye was entirely uninjured, although the scleral vessels were still engorged and cyanotically discolored when the eye was inspected.

The subsequent healing and convalescence were perfectly uneventful, and the final visual result obtained was excellent.

The following summary of symptoms due to cocain poisoning is taken from the *United States Dispensatory*, twentieth edition:

> There are two distinct types of cocain poisoning, one characterized by circulatory failure and the other by neurotoxic symptoms. The first type is usually seen after relatively small doses of the drug in persons who possess idiosyncrasy against it. The prominent symptoms are pallor of the face, vertigo, nausea, failure of the pulse and usually more or less complete loss of consciousness. These symptoms are supposed to be due to arterial spasm causing anemia of the brain. The other group of cocain poisoning is characterized by delirium, increased reflexes, more or less violent convulsions, the pulse usually being rapid and fairly strong, but later becomes weak, and syncope and cyanosis may intervene. The delirium is usually associated with hallucinations and at times the patient may develop a violent mania of even homicidal character.

(It is in this latter group that the case just reported should be classified.)

AN UNUSUAL ACCIDENT (BREAKING OF THE KNIFE POINT) OCCURRING DURING THE COURSE OF A CATARACT EXTRACTION

By HAROLD G. GOLDBERG, M.D.
OPHTHALMIC SURGEON TO THE HOSPITAL

As a warning to the inexperienced in ophthalmic surgery, and a protest against the carelessness of the makers of eye instruments, the following case should serve as an interesting example. At the time of selecting the von Graefe cataract knife for the corneal section, on numerous occasions the instrument has been rejected, because after returning from the instrument maker it was found to have a wire or spindle point. These knives will penetrate the testing drum with great ease, and will, therefore, being desirable in every other respect, perhaps deceive the operator. They are, however, a delusion and a snare, and if the point is further tested upon the finger nail or some other hard surface it will be found to have no stability at all but will bend one way or the other, so that it should be at once rejected as useless. If an attempt is made to penetrate the cornea with this knife the point will probably bend, generally following the layers of the corneal substance, and if the operator is so unfortunate as to accomplish this feat, he is almost certain to again turn the point at his counterpuncture, when he will find it necessary to either withdraw the knife, or, if the worst should happen, the point will break, with all the complications which might arise from this accident. Such has been the experience of the writer, as the following report will show:

A case for cataract extraction was prepared in the usual way, the instruments selected, and the knives, although there was a plentiful stock of them on hand, were found to be in rather poor condition. Finally one was found, however, which entered the drum with particular ease, and except that its point was somewhat slender, it was in every other respect worth selecting. It was looked upon with more or less suspicion, but was accepted as being the best that could be found. The circumstances of the case were somewhat peculiar, inasmuch as the patient had twice before appeared for operation and had been rejected because positive smears had been returned from the laboratory, where the conjunctival secretion had been sent for examination. While no attempt is made to excuse the operator on this ground, he was somewhat influenced by this condition of affairs, and while his better judgment dictated postponing the operation again, he was prevailed upon to proceed with it at once. The point of the knife was entered at the corneoscleral juncture in the usual position, and somewhat more than the usual amount of force was needed to complete the puncture.

At the counterpuncture the knife stopped and an assistant immediately exclaimed that the point had been broken, he being in a better position for the corneal reflexes to make this observation. The knife was carefully withdrawn, with very little loss of aqueous; a keratome was entered above as for a preliminary iridectomy and the anterior chamber opened without any further accident. Not wishing to use the large magnet, with its attendant dangers, and realizing the difficulty of grasping the fragment with the forceps, a search was made through the instruments at hand until one was found with sufficient magnetism (which had been acquired by use with the large magnet upon other occasions) to lift a medium-size needle. We were fortunate in finding an iris forceps to serve this purpose, and passing this instrument through the corneal wound an attempt was made to attract the fragment of steel. This was accomplished so rapidly that before the broken knife-tip reached the forceps it was drawn up behind the iris and entirely lost from view. An iridectomy was then performed and the section of iris tissue searched for the foreign body; it was not found, nor could it be seen anywhere within the anterior chamber. Believing that it might have slipped up behind the ciliary body the forceps were turned, point directed toward the ciliary region, and made to enter this region as far as possible without injury to the part. Upon the removal of the forceps the steel fragment was found clinging to the instrument by its magnetic influence. The eye was dressed in the usual manner, the healing was uneventful, and the result a preliminary iridectomy instead of a combined extraction, as had been at first planned.

A CASE OF GLIOMA RETINÆ, WITH INVOLVEMENT OF THE FELLOW EYE FOUR WEEKS AFTER ENUCLEATION

By DAVID J. BOON, M.D.
ASSISTANT OPHTHALMIC SURGEON TO THE HOSPITAL

The object of the writer in reporting this case, aside from the general interest manifested in these neoplasms of unknown origin, with such rapid and destructive growth, is twofold:

1. To show how negligent parents may be in securing for their children proper medical attention.

2. To show how a careless diagnosis in an eye dispensary can be followed with such deplorable and fatal results.

The family history of the case is as follows:

Marie W., aged three years, American. The mother and father are both living and in good health. There is no history of cancer or other neoplasms in either family. There are two brothers and one sister in good health and not any of these children have any eye affection. There was nothing unusual at the time of the patient's birth; it being a normal labor of moderate duration; without instruments.

Present Medical History. The child's eyes were normal at birth. At the age of one year the mother noticed a small white spot in the left eye, which she felt was not of sufficient importance to consult anyone about until one year later, when she took the child to an eye dispensary, where a diagnosis of congenital cataract was made and non-interference advised. This plan was followed for about four months, with the eye gradually getting worse, until finally the child was brought to our dispensary, under Dr. Goldberg's service, on October 29, 1919, and the following conditions of the left eye were noticed: The entire vitreous chamber was filled with a mass presenting a yellowish-white reflex; the crystalline lens was pushed forward against the posterior surface of the iris and the entire anterior chamber was obliterated. There was considerable scleral injection, some pain, increased tension and total loss of vision. An examination of the fundus of the right eye was negative and the retina appeared to be perfectly normal. The diagnosis of glioma retinæ was made and an immediate enucleation advised, which operation was performed on November 3, 1919. The optic nerve, which was sectioned as deeply posterior as possible, was found to have a peculiar translucent appearance slightly pinkish in color, not presenting the typical coloring of a normal nerve. The specimen was sent to the laboratory for examination and the diagnosis of glioma retinæ confirmed by the pathologist. The patient had an uneventful convalescence but was removed from the hospital by her father against our advice after signing a release on November 8, 1919, only five days after the operation. She returned to the dispensary November 15, 1919, when the following observations were made: The right eye was negative. In the left eye there was some discharge but the socket conjunctiva was flat, smooth and about as clean as is usually found in such cases. November 22, 1919 she was again examined in the dispensary; and found to be progressing favorably, there being no signs of recurrence. December 2, 1919, she was again brought to the dispensary, and her mother stated that she complained of pain in the fellow eye. An examination of the fundus of this eye revealed beginning involvement of the retina just four weeks after the enucleation. December 6, 1919, she still complained of pain and a grayish reflex could be distinctly seen; enucleation of this eye was advised but the parents refused and decided to consult someone else. This latter course was followed, and having received the same advice as ours, they decided not to have the child operated. They returned to the dispensary February 21, 1920, seeking relief for the child's pain. The vitreous chamber of the right eye was almost entirely filled with the mass by this time but the lens was still in good position. Again an operation was suggested and was refused, the parents preferring to sacrifice the child's life rather than have her go through life totally blind.

CONCLUSIONS. 1. The consulting with a skilled oculist early in the course of this condition would have in all probability saved both this child's right eye and also its life. The parents erred, as many of them do, in not seeking medical assistance until it is often too late to accomplish the most desirable results.

2. The differential diagnosis between congenital cataract and glioma retinæ should be familiar to every ophthalmologist. These congenital lens defects along with inflammatory deposits in the vitreous chamber, whether the retina may or may not be detached, should be grouped under the heading Pseudogliomata. All such cases appearing in dispensary practice, where there is the slightest doubt as to the diagnosis, should be studied most carefully with an ophthalmoscope, with the pupil properly dilated, and if the child resists, as many of them will, chloroform should be administered so that a most thorough examination can be made. By this method only is a very valuable physical sign obtained: the tension of the eyeball, which is found to be much increased in glioma and lowered in all forms of pseudoglioma. If after a careful examination the slightest doubt still exists the eye should be removed because the child's life is at stake and the eye will probably always be sightless and somewhat of a menace at the best.

LETHARGIC ENCEPHALITIS, WITH A REPORT OF SIX CASES

By ARTHUR H. HOPKINS, M.D.
PHYSICIAN TO THE HOSPITAL

AND

WILLIAM B. SIMS, M.D.
RESIDENT PHYSICIAN IN THE HOSPITAL

In view of the prevalence of this disease in the train of the epidemics of influenza during the past two years and in view of its many and varied features, it has seemed to us worth while to present briefly the cases we have recently had on the Men's Medical Service in the Hospital.

Various types of encephalitis have been given, but probably the best classification to date is that of McNally, which is as follows:

1. Cases with general symptoms and without localizing signs.
2. Cases with third nerve paralysis and general disturbance in the function of the central nervous system.
3. Cases with facial paralysis and general disturbance in the function of the central nervous system.
4. Cases with spinal manifestations and general disturbance in the function of the central nervous system.
5. Cases with polyneuritic manifestations and general disturbance in the function of the central nervous system.
6. Cases with mild or transient manifestations (so-called "abortive" cases).

To these should be added cases of paralysis of other motor cranial nerves than the third and fifth, such as those of deglutition and respiration.

The symptomatology of the disease is both broad and inconstant, as will readily be exemplified by the small group of cases here presented.

Owing to this fact we may realize that, to the practising physician who has seen few if any of these cases, the unusual types may present some difficulty in diagnosis.

CASE REPORTS

CASE I.—J. G., aged forty-five years, a grocer, was admitted to the Episcopal Hospital April 15, 1920. The family history was negative. His previous history was negative until two months before admission, at which time a cough, slight mucoid expectoration and loss of weight and strength began. During the past two months he has felt indisposed for work—rather tired and sleepy.

About four weeks before admission the general weariness and sleepiness increased. He continued work for two weeks longer, when he went to bed, complaining of exhaustion. Following this his wife noticed that he began acting queerly and talking in a rather peculiar way. He was put to bed but refused to stay there. He was most irrational at night and slept very little, due to dreams, and finally, the day before admission, he went into a restless, muttering delirium.

Examination on admission showed a rather anemic man with masked facies and a toxic appearance. He was restless and refused to stay in bed; he had slight difficulty in speech, talked incoherently to himself, but would answer questions intelligently, only to drift back into mental confusion.

The positive physical findings were as follows: Pupils were contracted and reacted sluggishly to light; the tongue was tremulous on protrusion; there was general muscular hypertonia, especially of the neck, but no actual rigidity; normal abdominal reflexes but exaggeration of knee reflexes, and a positive Kernig's sign on both sides.

After three days, from a condition of restless, muttering delirium (in which restraint was necessary), he lapsed into a deep coma. He lay with mouth open, eyes closed and facies immobilized, and did not respond to questions. During the three days he ran a hectic temperature, ranging from 97° to 103° (rectal), as will be seen on

Chart I. He took liquids well and his excretory functions were normal. During the next three days he steadily became worse, developing ptosis, paralysis of the soft palate, dropping of the lower jaw and finally death (April 21, 5 A.M.) from respiratory paralysis.

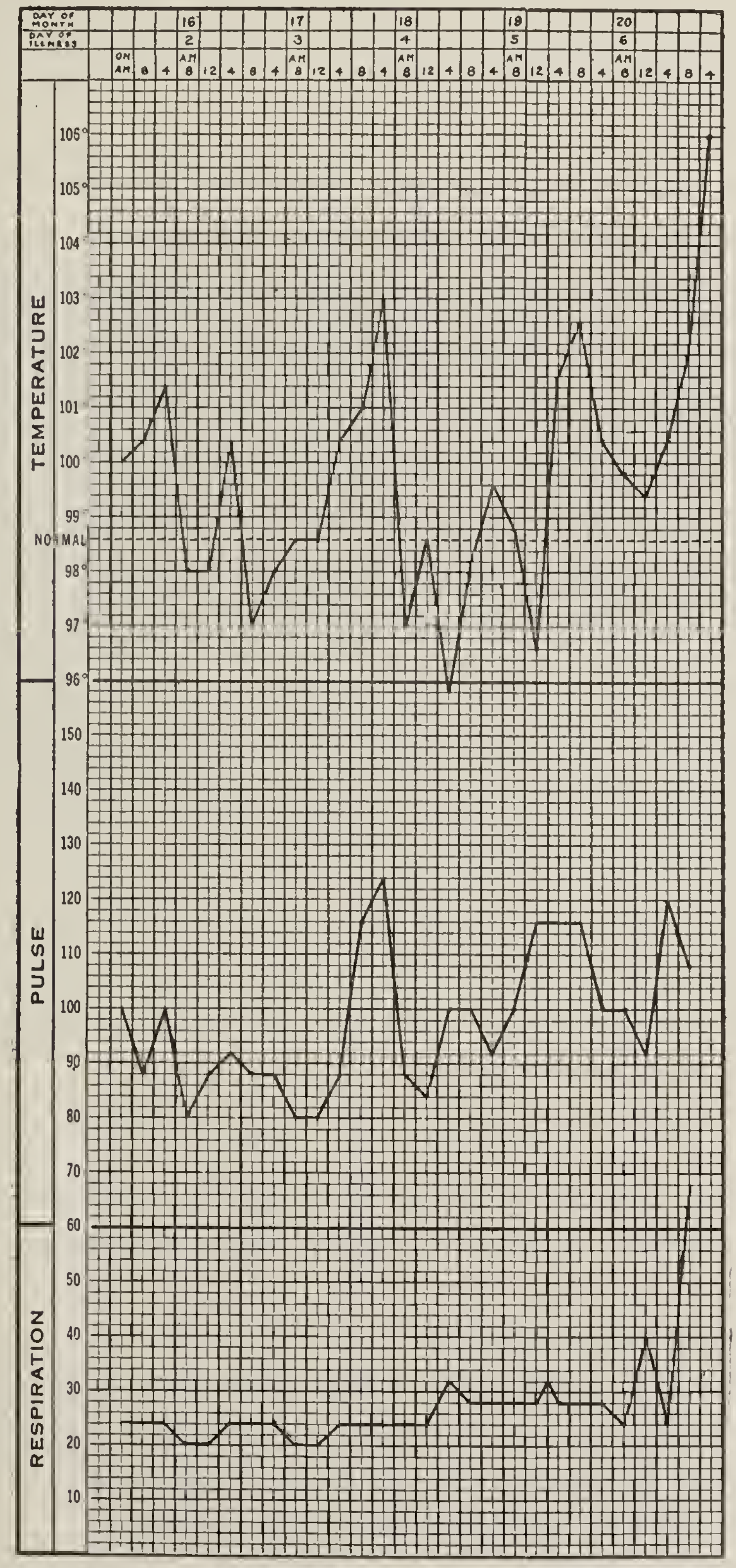

Chart I.

Laboratory Findings: Urine: Specific gravity, 1020; cloud of albumin; granular casts.

Blood: White blood cells, 11,600; red blood cells, 4,300,000; hemoglobin, 65 per cent.; polymorphonuclears, 62 per cent.; mononuclears, 12 per cent.; small mononuclears, 24 per cent.; basophiles, 2 per cent. Wassermann, + + +.

Spinal fluid: Not under pressure, clear, 91 cells per c.mm.; polymorphonuclear, 90 per cent.; lymphocytes, 10 per cent.; Fehling's reduced; globulin negative.

Remarks. The interesting features in this case are the absence of any previous apparent influenzal infection aside from slight bronchial irritation, the absence of the more usual initial symptoms, as diplopia, ptosis, etc., and, finally, the severe maniacal development which ensued. How far the syphilitic infection influenced this case is difficult to say.

The prodromal period may be regarded as having existed possibly for a period of two months. Fever may or may not have been present during the prodromal period, but while he was in the hospital he ran a very hectic temperature, ranging from 96.4° to 106°, the latter being terminal. The presence of a Kernig's sign is of particular interest, in that it is usually described as being lacking in this condition. We must also note the rather high cell count in the spinal fluid (91), although it is by no means uncommon to find a count as high as this. We are inclined to place this case in the psychosis group of Tilney and Riley.

Case II.—C. F., aged forty-three years, was admitted to the Episcopal Hospital April 16, 1920, the diagnosis being undetermined.

Family history was negative.

Personal History. Negative until four weeks before admission, when he had influenza followed by rhinitis and pain in the frontal sinus and in the left ear. These cleared up in a few days and he felt well until the beginning of the present illness.

Present Illness. Began one week before admission, when he developed pain in the left shoulder, muscles of the chest and in the gastric region. Four days before admission he developed tremors of the hands and twitching of the abdominal muscles and lower intercostal muscles, associated with shooting pains over the same

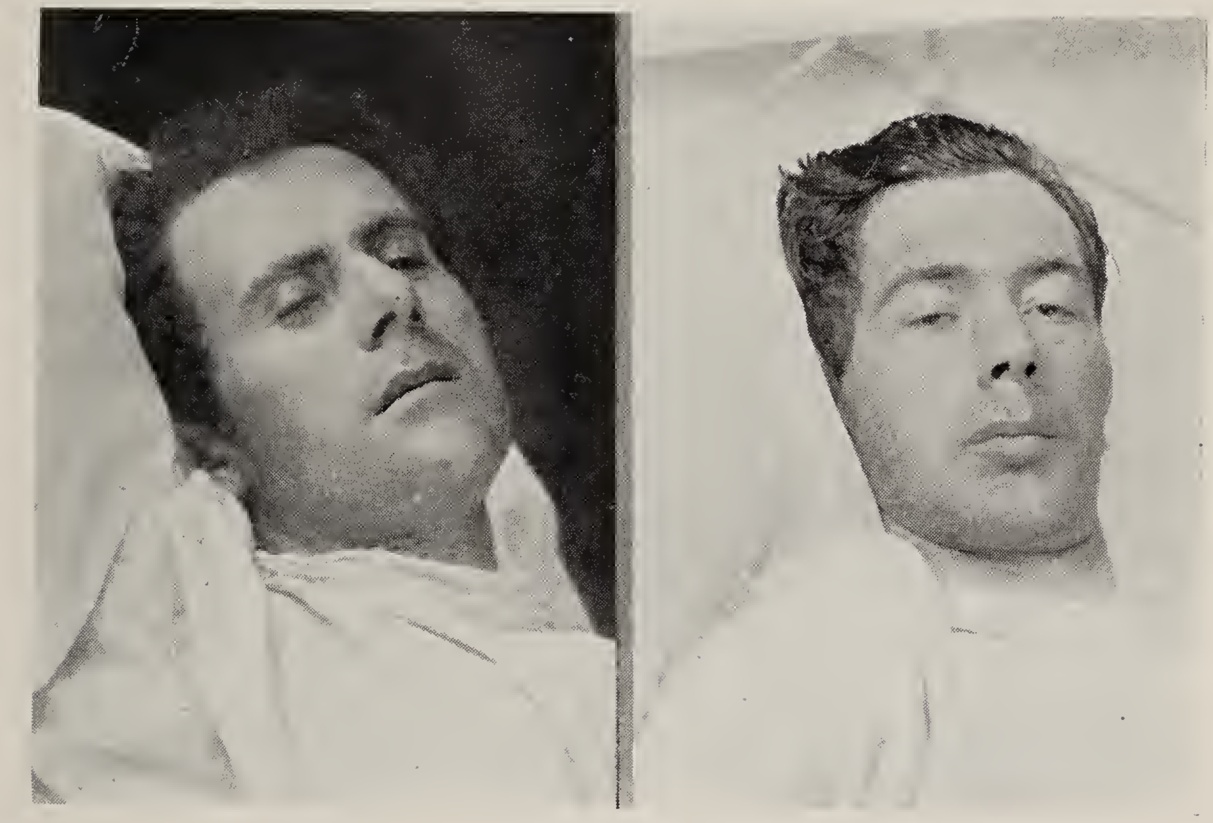

FIG. 106.—Case 1, J. G. and Case 4, W. T.

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region. He was unable to sleep for three nights before admission, due to the constant contractions of the abdominal muscles, associated with shooting pains.

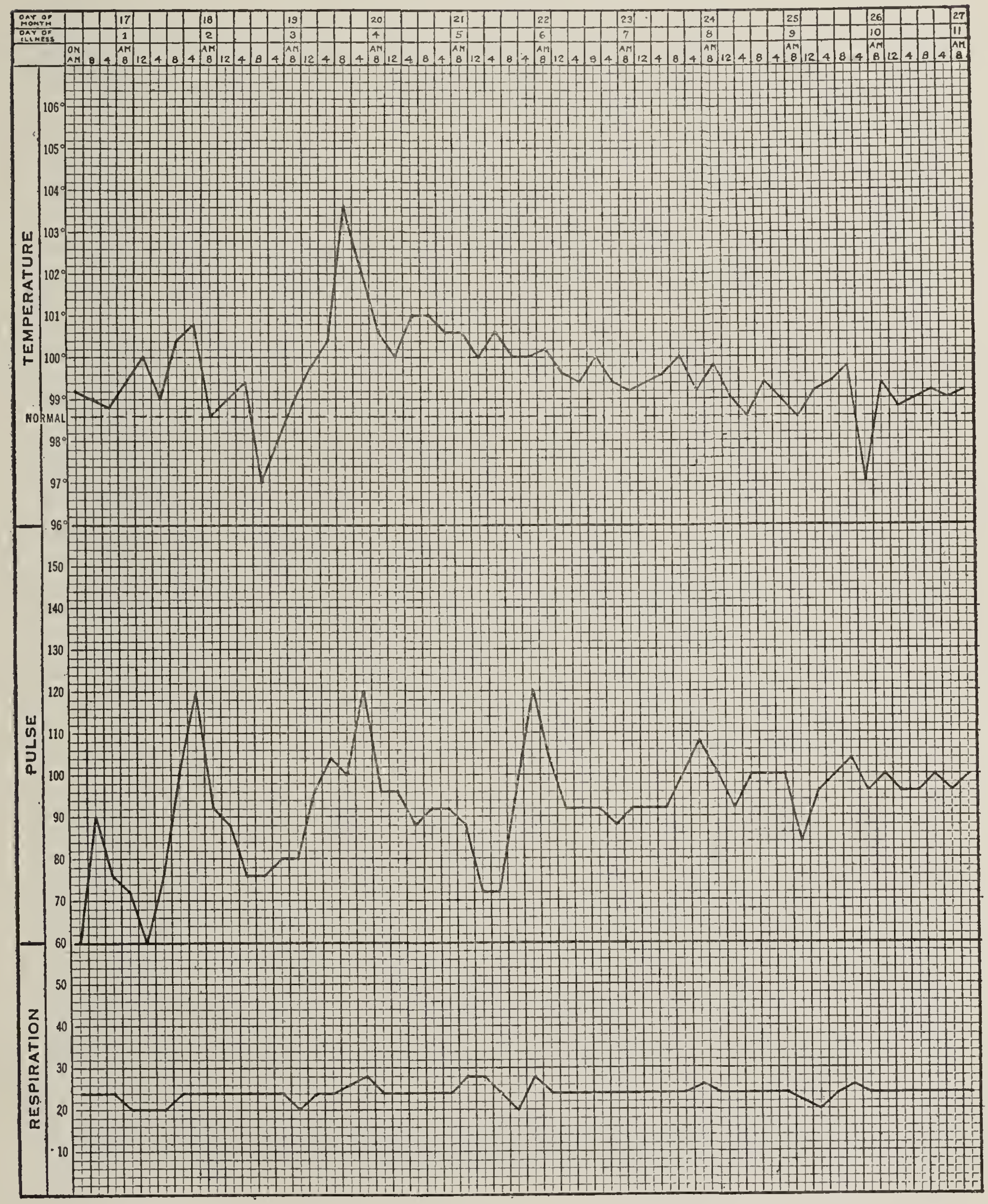

Chart II.

On examination there were no visionary disturbances or palsies. Expression somewhat masked. Heart and lungs presented no abnormality. The abdomen was distended and the upper recti muscles were slightly rigid; some tenderness to pressure over the epigastrium. There was a constant clonic contraction of the upper abdominal muscles, the lower intercostals and apparently the diaphragm. He had considerable belching but no hiccoughs, nausea or vomiting. Abdominal reflexes were present, as were also the knee reflexes. Kernig's sign was negative, no clonus or Babinski. Slight tremors of the hands. The mental condition was lucid.

Notes on Patient's Condition while in the Hospital. The patient talked a great deal during the night, got out of bed and seemed confused when put back. During the following five days he was irrational at times and had delusions and hallucinations and slept but little.

Abdominal twitching was constant. Temperature ranged from 100° to 103°, as will be noted on Chart II. From this state of delirium, in which restraint was necessary, he became lethargic, but could be aroused and answered questions rationally. At the end of five days more, although he slept a great deal, there was marked improvement; his expression was more natural and for the first time the abdominal twitchings were absent. He complained of a tingling and numbness of the skin and shooting pains in the chest and abdomen. These gradually disappeared, as did all tremors, but lethargy was marked for a week following, during which time the patient often fell asleep while sitting up.

The patient eventually recovered.

The condition in this case clearly followed an influenzal infection of recent origin. From the history it may readily be seen that this patient falls more within the polyneuritic classification, though the pronounced maniacal tendency rather precludes a clear-cut classification. On admission to the hospital the spasmodic contractions of the muscles of the upper abdomen were violent and very frequent; this symptom, though very pronounced in this patient, was entirely lacking in the others. By some it has been regarded as pathognomonic of lethargic encephalitis.

Here, again, we note the absence of diplopia, blurred vision, photophobia and other localizing symptoms. His temperature throughout his stay in the hospital was somewhat hectic, ranging from 96° to 103.3°.

CASE III.—S. B., aged fifty-two years, was admitted to the Episcopal Hospital April 14, 1920.

Family History. Negative.

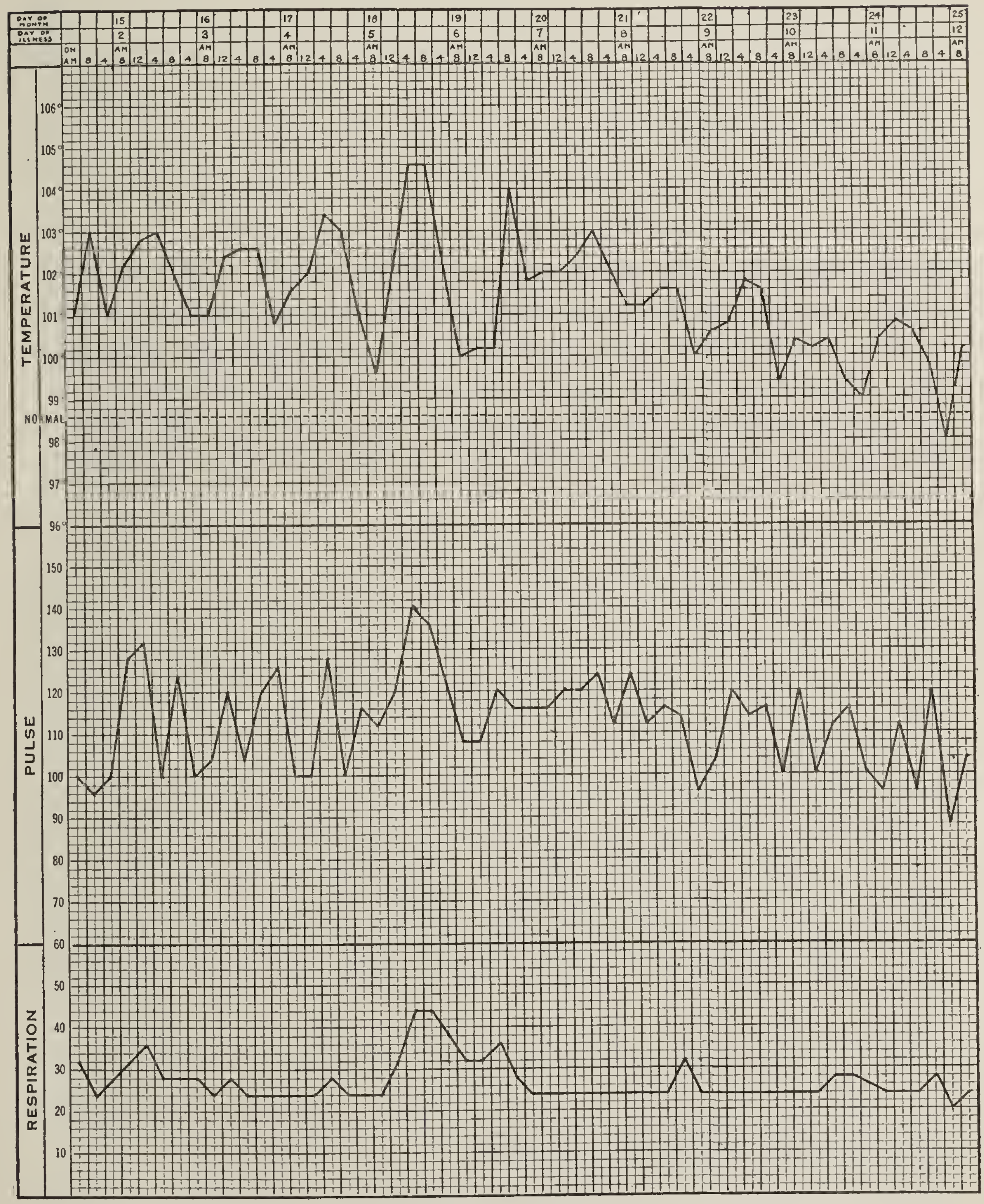

Chart III.

Personal History. About two months before admission the patient began to suffer with nervousness, "gas on the stomach" and epigastric pain. He also had pain in the muscles of the legs with tingling and numbness.

Present Illness. The present illness is a continuation of the above symptoms. The tremors of his legs have become more persistent and violent, and he has felt nervous and unable to sleep.

Examination showed an extremely restless and nervous man. There was slight difficulty in speech, with stuttering and scanning. Facies masked. There was a suggestive bilateral ptosis; fundus oculi negative. The pupils were contracted and equal, responding but slightly to light reflex and to accommodation. The tongue was tremulous on protusion and deviated toward the right. No abnormality of the heart or lungs. There was muscular resistance of the abdomen, and the reflexes were present, and a tâche was noted. The tremors of the legs were convulsive and the knee reflexes at this time could not be elicited. Ankle-clonus was marked on the right side, Babinski was negative and Kernig's sign suggestive. There were marked tremors in both hands.

Notes on Patient's Condition while in the Hospital. The patient went into a muttering delirium and became somewhat resistive, rendering restraint necessary. He slept some of the time but muttered almost constantly. After three days of restless delirium, during which time his temperature rose from 101° to 104°, (as will be seen on Chart III), he went into a profound lethargic state and could be aroused only with difficulty. His tremors at this time disappeared and the knee reflexes were present. Marked bilateral ptosis, difficulty in speech, masked facies and lethargy were most pronounced at this time.

During the following ten days there was a gradual fall in his temperature from 104° to normal, and gradual improvement in his mental and general condition. He slept most of the time quietly, but occasionally got out of bed and seemed disoriented. All his symptoms gradually disappeared, but during his whole illness he complained of being tired. He made an uneventful recovery.

Laboratory Findings. Urine: Showed a trace of albumin and occasional granular and hyaline casts.

Blood: Leukocytes varied from 14,000 to 11,000. Differential count normal. Wassermann negative.

Spinal Fluid: Fehling's solution was reduced; globulin negative; 45 cells per c.mm. Wassermann negative.

Aside from a possible prodromal period from January 1 to February 22 there were no symptoms other than excessive nervousness. The evidence of polyneuritis, high fever and pronounced maniacal tendency are found here to be the chief characteristics of the case, and owing to the lack of further early symptoms the diagnosis at first was in doubt; but later the pronounced ptosis of the eyelids, the masked facial expression, along with the mania and later the lethargy, made the diagnosis become very clear. The temperature was hectic, fluctuating from 96° to 104.3°. Once again we note a positive Kernig's sign and a rather high cell count of the spinal fluid (45). Again in this case the classification, as in Case II, we believe should lie between the polyneuritic and psychosis types, though the symptoms were of such a character as to render a clear-cut classification difficult.

CASE IV.—W. T., aged thirty-two years, was admitted to the Episcopal Hospital April 6, 1920.

Family History. Negative.

Personal History. Negative until two months before admission, at which time he had influenza, which confined him to bed for one week. Following this he had a frontal sinus infection which cleared up in a few days.

Present Illness. Three weeks before admission, after feeling unusually well, the patient awakened one morning with diplopia. This continued for one week, when he consulted an "eye specialist," who dilated and refracted the eyes, but the diplopia continued and a purulent conjunctivitis developed. One week before admission he went to bed, complaining of his eyes and of being drowsy. Four days before admission he began having difficulty in articulation, which has increased, until at present the patient cannot speak. The day before admission he had difficulty in swallowing. During the week that he has been in bed he has been quiet and drowsy and has made no complaint. For several days muscular twitchings around the mouth have been present.

Physical Examination. The patient lay in bed with eyes half-closed, in a lethargic state. His pupils were dilated and did not react to light; there was bilateral ptosis. The fundus was hazy and there was slight swelling of the nerve head, with edema of the retina and engorgement of the veins. Catarrhal conjunctivitis was present. No intra-ocular palsies. The face was expressionless and there were tremors of the muscles of the mouth, most marked when the patient was disturbed. He could not talk, though he understood and made an attempt to coöperate. The tongue, which was protruded with difficulty, was tremulous and coated. There was resistance in the muscles of the neck but no rigidity.

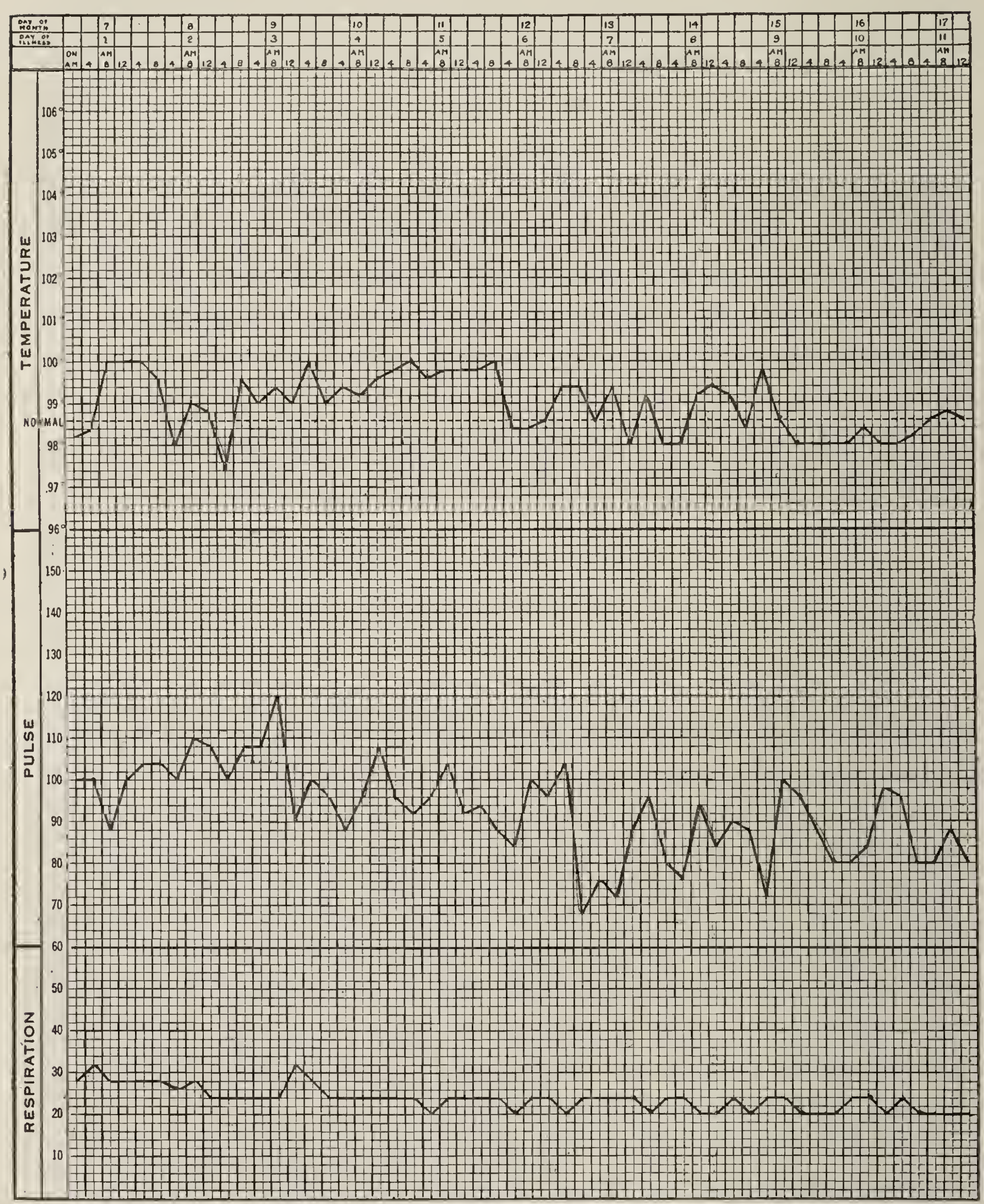

Chart IV.

Heart, lungs and abdomen were negative. Reflexes present. Knee reflexes were increased; no Babinski nor ankle clonus; Kernig's sign was suggestive. There was general muscular hypertonicity.

Laboratory Findings. Urine: Negative.

Blood: Leukocytes, 9400; differential count normal; red blood cells, 4,150,000; Wassermann negative.

Spinal fluid: Cell count, 23 cells per c.mm.; polymorphonuclears, 85 per cent.; Fehling's reduced; globulin negative. Wassermann negative.

Notes on Patient's Condition while in the Hospital. Patient was lethargic, without apparent cause, for eight days. He swallowed liquids fairly well. Twitching of the mouth muscles was constantly present; the facies was masked and muscular resistance general. At the end of a week the conjunctivitis cleared up and he began to talk slowly and showed general improvement. At the end of three weeks (April 26, 1920) he was able to articulate distinctly and felt well, although tremors were still persistent.

April 27, 1920, following a prolonged visit of some of his friends, his condition did not seem so satisfactory. He again developed great difficulty in speech and in swallowing, and stared at one object constantly with eyes wide open. He reacted slowly. Complained of tingling and numbness of the third and fourth fingers of each hand and his sleep was disturbed by dreams, and he, for the first time, seemed solicitous about his condition.

At the present time the patient looks fairly well but there is no marked improvement in the last few weeks. Although lethargic in appearance he sleeps but little. He has always been rational. His temperature has never been above 100°, as will be noted on Chart IV, but there has been slight fever most of the time since the onset of the illness, with occasionally a few days of intermission.

Recovery was slow and uneventful.

Case V.—C. L., aged twenty-two years, was admitted to the Episcopal Hospital April 30, 1920.

Family History. Negative.

Personal History. Has suffered from rheumatism off and on for several years. Twelve weeks before admission the patient had a severe attack of influenza, followed by double pneumonia and pains of a rheumatic character in the shoulders and legs. He was confined to bed for eight weeks. The following two weeks, while he recuperated in his home, he suffered from a severe cough, with slight expectoration. During this time he complained of weakness, exhaustion and dizziness, and slept a great deal. He also complained of rheumatic pains in the shoulders and legs.

Two weeks before admission he returned to his work driving a milk wagon, and at the time complained of being sleepy. He would sleep thirteen hours a day and complained of inability to wake up. During the last two weeks he has seemed rather irritable and his speech has been unnatural. On the morning of his "stroke" he complained of seeing double and being unable to change money properly.

Present Illness. Four days before admission, while driving a milk wagon, he suddenly lost his speech. He drove back to his home and after entering had a sudden collapse on a sofa. At this time his expression was unnatural and he had general tremors, most pronounced in the legs. His wife thought he was having a severe chill. Following this he was unable to move his left side and his speech did not return. After four days at home, during which time there was no change in his condition, he was admitted to this hospital.

Examination on Admission. Well-developed man, with masked expression, markedly jaundiced and unable to talk or move the left arm or leg. Eyes reacted to light and accommodation; no nystagmus or ocular palsies; sclera slightly icteric; eye-grounds negative. The mouth seemed to be pulled toward the right and he was unable to move the facial muscles of the left side and unable to wrinkle the forehead. There was a slight tremor of the tongue on protrusion. Patient heard perfectly and coöperated when it was possible. The neck showed slight muscular resistance. Chest negative except for a few moist rales at the base of the right lung. Heart shows forcible pulsation against the chest wall; precordial thrill, blowing systolic murmur heard most distinctly over the pulmonic area. First sound loud and roughened. Quality of sounds poor and varied a great deal over the different areas; second aortic sound accentuated; marked arrhythmia; slight enlargement of the organ. The abdomen showed marked jaundice and slight tenderness over the gall-bladder region, with muscular resistance of the upper abdomen. Reflexes normal, tâche obtained. Bladder not distended.

Extremities: Knee reflexes present and exaggerated on the left side; Babinski present on the left side, suggestive Kernig's on both sides, flaccid paralysis of the left arm and leg.

Notes on Patient's Condition while in the Hospital. The patient has been in the hospital just one week. During this time he has improved in every way. He can articulate fairly well and speak short sentences. Facial paralysis still marked on the left side. He has had perfect use of the previously paralyzed side. He eats well and sleeps normally. The jaundice is lessening. He complains of epigastric pain occasionally. His mental condition is clear.

His temperature is normal and he seems to have a fair chance to make a complete recovery.

Later note: Recovery was complete.

CASE VI.—T. B., aged thirty-four years, an Italian, was admitted to the Episcopal Hospital May 10, 1920.

Family History. Negative.

Personal History. Negative until March 11, 1920, at which time he had influenza and was in bed for about two weeks. Following this he had a pain in the right ear and over the right eye, which passed off in a few days, although hearing in his right ear has been impaired ever since. During the past six weeks he has been recuperating in his home and felt quite well until two weeks ago.

Present Illness. Two weeks before admission he began feeling badly but he had no definite complaint. His family physician suspected beginning typhoid fever. Following this feeling of malaise and anorexia he began having twitching of the muscles of the neck on the right side, later becoming associated with sharp pains, neuritic in character, in the neck and shoulders and down the right arm. Since the beginning the muscular twitching has been constant and has become more or less convulsive in character, and the pain has become severe enough to keep him awake at night. Six days ago he began having tremors of both hands and also diplopia, lasting two days. Four days ago he began having difficulty in articulation. His chief complaint on admission was twitching of the muscles of the neck, pain in the neck and right arm and inability to sleep.

Examination on Admission. Facial expression masked. Head retracted backward and slightly to the right side. Pupils dilated but respond to light and accommodation. Eye-grounds negative. Marked tendency to stare. Slight impairment of hearing in the right ear. Tongue protrudes well in the midline and is not tremulous.

Neck: Constant convulsive twitching of the trapezius muscle, most marked in the cleidoöccipitalis cervicalis portion. There was the painful sensation of an ordinary neuritis, confined to the nape of the neck, shoulder and right arm and hand. There was marked muscular resistance of the neck.

Chest, heart, lungs and abdomen negative.

Extremities: No Kernig's sign; knee reflexes increased; no ankle-clonus; no Babinski.

Laboratory Findings. Blood: Erythrocytes, 5,890,000; leukocytes, 8200; hemoglobin, 75 per cent.; Wassermann negative.

Spinal fluid: Cell count, 21; Fehling's reduced; globulin negative; Wassermann negative.

Urine: Negative.

The patient has at the present time been in the hospital just one week. His temperature has been normal and his mental condition clear. He has had very little sleep, due to the pain in the neck and arm and hand. He has developed partial ptosis and the tendency to stare, and the masked expression is more marked. The twitching of the right trapezius has been constant, and there is marked quivering of the muscles of the arms and tremors of the hands. He coöperates very slowly and articulates with a great deal of difficulty.

Physical examination otherwise is the same as on admission.

The patient eventually recovered.

SUMMARY. As noted in the first part of this paper the variety of symptoms and the absence of some of the most characteristic ones may present diagnostic difficulties to the practising physician.

The prodromal period in these cases was variable and a history of influenza as a precursor was present in four and absent in two.

CASE I.—The onset of the first case was slow (two months) and was characterized by upper respiratory irritation, lethargy and fatigue. The severity gradually increased until at its height mania and high fever were very pronounced.

Case II also presented a slow onset, following influenza and later a rhinitis. His symptoms were ushered in by pain in the shoulders, chest and gastric region and tremors of the hands and abdominal muscles. Probably of all of his symptoms the most pronounced and most constant were the persistent contractions of the abdominal muscles, though the mania and high fever were also very marked. Diplopia and some of the other characteristic symptoms were lacking.

Case III presents a prolonged prodromal period, characterized chiefly by nervousness, followed by mania, pain, twitching in the muscles and high fever. The delayed development of some of the more characteristic symptoms presented some difficulty in diagnosis in the earlier stage.

Case IV was probably more typical of the many cases presented in the literature and offered no difficulty whatever from the viewpoint of diagnosis.

Case V.—Here, again, influenza may be regarded as a precursor of the disease, which latter was ushered in by lethargy, vertigo, exhaustion and then neurotic pains in the shoulders and legs, all of which were sufficiently suggestive to warrant a tentative diagnosis of encephalitis, a diagnosis confirmed by the development of diplopia, loss of speech, masked facies and then hemiplegia. Jaundice as a complication was unusual as was the hemiplegia, the latter being regarded in the literature as a very rare occurrence.

Case VI.—Preceded by influenza and ushered in by malaise and twitching of the muscles of the right side of the neck, followed by neuritic pains in the shoulders and arms, diplopia, etc., the case presented no difficulty in diagnosis. The intense and persistent rhythmic contraction of the trapezius stands out as the striking feature here.

Five of the six cases recovered.

A CASE OF LETHARGIC ENCEPHALITIS WITH PRIMARY MASTOIDITIS

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AND

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The case we are presenting emphasizes the necessity of ever being on guard; now that epidemic encephalitis is the topic of the day, one is liable to jump too hastily into the diagnosis and overlook some very important conditions.

The diagnosis of encephalitis lethargica could very well have explained all the symptoms of our case, including the pain and tenderness in the left postauricular region, which later proved to be an entirely independent condition, namely, a primary mastoiditis.

William House, in the *Journal of the American Medical Association*, February 7, 1920, reports a case illustrating our point: "Throughout the illness he complained of pain in the back of the neck and back of the left ear, which was deep seated and at first thought to be due to neuritis of the occipital group of nerves. After the beginning of the apathetic stage his pain became more troublesome, making him restless at night and he was sensitive to touch throughout this region." We are presenting this case as unusual for its peculiar coincidence of lesions.

F. K., a boy, aged sixteen years, poorly developed and poorly nourished, occupation a factory worker, was admitted to the Episcopal Hospital on the evening of March 17, 1920, with a history as follows: On Monday, March 15, the patient had a spasm of coughing, which was of over three hours' duration, finally relieved by some pills given by a physician; that night the patient was unable to sleep, the reason given being nervousness. On the following day, Tuesday, he had a pain in his epigastrium, and again on Tuesday night he had no sleep despite drugs administered by his physician. On Wednesday morning pain behind both ears started and later in the day he was sent to the hospital. Later, on close questioning, the patient admitted having had diplopia previous to the onset of his illness. We saw the boy on Wednesday, March 17, 1920, at 11 P.M. The patient was extremely restless, with continual tossing of his body, limbs and head in an incoördinated way, best described by the term exaggerated choreiform movements. When spoken to the patient would reply promptly and intelligently, and when commanded to hold still he would remain quiet for a short time.

On examination he was tender over both mastoid regions and was extremely hyperesthetic in all parts of his body, especially in the left postauricular region and left sternocleidomastoid muscle, which stood out, as did the right, in tonic spasm. The generalized hyperesthesia and choreiform movements made local symptoms difficult to determine definitely at this time.

There was spontaneous horizontal and vertical nystagmus present; the eyeball movements were not synchronous with the muscle twitchings as observed by Dr. C. J. Swan in one case.[1] The external auditory canals were normal, each membrana tympani normal, as was the hearing to both watch and voice tests. There was a marked mucopurulent rhinopharyngitis present and the tonsils were inflamed, enlarged and cryptic, being bathed with a profuse secretion from the nasopharynx. The tongue was dry, fissured and contained heavy brownish crusts; the lips were cracked and the teeth presented sordes. Lumbar puncture had been performed; the spinal fluid was contaminated by the admixture of blood, but the pressure was normal, dropping about fifty drops per minute. Two subsequent lumbar punctures, one at the time of operation on March 18, 1920, and the other six days after operation, were both negative, with a cell count of eight and six respectively to a cubic millimeter. Globulin tests were negative. The temperature on admission to the hospital on March 17, 1920, was 101.3°, pulse 92, and respirations 24. Leukocyte count was 15,000. Babinski normal. Ankle-clonus was absent. The neck showed no rigidity except for spasm of the sternocleidomastoid muscles. Kernig's sign was suggestively positive.

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[1] History report of cases by Dr. Peter Bassoe in the Journal of the American Medical Association, April 10, 1920.

Our opinion at this time was that the patient had a basal meningitis, toxic or infectious, and that the portal of entry to the brain was probably by way of the nasopharynx or sinuses, because of the marked degree of inflammation present. The mastoid was disregarded because of normal hearing and normal membrana tympani, attributing the mastoid tenderness to the general hyperesthesia.

The following afternoon, March 18, after the patient had been quieted somewhat by morphin sulphate, grain $\frac{3}{4}$ hypodermically, sodium bromide, grains xxx, and chloral hydrate, grains x, per rectum, administered on admission, the latter being repeated in eight hours, we saw the patient again. The hyperesthesia had markedly decreased, but very slight if any change was noted in the intensity of the choreiform movements. There was definite tenderness on light palpation over the left mastoid region but the tympanic membrane remained normal.

The *x*-ray report (Dr. R. S. Bromer) was: "There is a definite loss of cell outline and haziness throughout the left mastoid. This is suggestive of involvement, due either to pus or serum."

Eye-grounds examined and found negative.

As the patient was very toxic and was complaining of severe headache and pain behind the left ear, and because of the localization of tenderness, with the positive *x*-ray findings, we decided to do an exploratory mastoid operation.

The operation was done at 10 P.M. on March 18, the usual mastoid incision being made. There was no thickening or adherence of the periosteum. The mastoid cortex appeared normal. After removal of the cortex we discovered a diffuse rarefying osteitis extending from the tip of the mastoid and external antral wall to the tegmen tympani. The mastoid process was exenterated of all necrosed bone and probed for sinuses in the tegmen tympani and attic, with negative findings. The wound was closed and drained in the usual manner. No incision was made in the tympanic membrane.

After the patient came out of ether the choreiform movements returned, his restlessness was still marked, but he was relieved of his pain and headache. The choreiform movements remained until March 20, two days after the operation, and then a gradual improvement in his condition began. With the cessation of the restlessness the patient seemed to sink into an exhausted state bordering on stupor, which became more pronounced until he developed a slight delirium. Most of the time he was apathetic or lethargic, awakening only by commands and to receive nourishment; at times he would try to get out of bed and had to be restrained. After three or four more days his delirium subsided, his lethargic state also gradually cleared up; by March 29, eleven days after the operation, he was almost free from his stupor. His temperature, which ranged between 100° to 103° F., descended by lysis and remained normal after April 6, nineteen days after operation. There was no paralysis of the central or peripheral nerves, excepting a suspicion of hemiparalysis of the tongue, which deviated to the right on protrusion. Deglutition was normal.

For three weeks the patient complained of pain in the extremities. The spontaneous nystagmus subsided entirely on the eighth day after the operation.

The patient is now convalescing, with no signs or symptoms excepting weakness. His hearing is normal to watch and voice and the mastoid wound is entirely healed.

Comments. The diagnosis of mastoiditis was confirmed by the operative findings and the relief of symptoms after operation. As to the diagnosis of epidemic encephalitis, we refer to the report of a similar case by Dr. Peter Bassoe, in the *Journal of the American Medical Association*, April 10, 1920, where he describes symptoms referable to severe acute chorea, and as a new symptom he gives his observation of the twitching of the abdominal muscles. Cases with pain in the extremities Dr. Bassoe classifies with a type called the "meningoradicular type."

Dr. Flexner, in his article of March 27, 1920, in the *Journal of the American Medical Association*, says: "Symptoms referable to irritation of the meninges may be present, usually slight, and while sometimes arousing suspicion of acute meningitis, that condition is excluded by lumbar puncture and examination of the cerebrospinal fluid. In addition, the important point of the rare presence of Kernig's sign should be mentioned."

Attention is again called to the fact that our patient had a suggestive Kernig's sign.

Dr. I. H. Jones says that vertical nystagmus is pathognomonic of pontocerebellar angle lesion, inflammatory or non-inflammatory. We had it present here, due probably to the meningeal irritation of some undetermined cause, possibly toxic.

The delirium and later the lethargic state, the characteristic fever and laboratory findings, together with the abovementioned clinical symptoms, all point to the diagnosis of encephalitis lethargica, coming under Bassoe's meningoradicular type.

The interesting feature of the case is the totally uncomplicated recovery. We are certain that an early mastoidectomy was indicated. We are not certain as to the contribution of the lumbar punctures to his recovery; moreover, these were performed more for diagnostic purposes, but there is a possibility that the withdrawal of spinal fluid relieved some of the toxemia, even if it was employed empirically as in any unknown form of meningitis.

Other points of interest are normal hearing and a normal drum membrane, with a diffuse subcortical mastoid osteitis.

The patient, although delirous at times and apathetic most of the time during the course of the disease, presented a mental state which seems almost incredible when one considers the gravity of his physical condition, in that he remembers most of the course of his disease, prodromal symptoms and his entrance into the hospital.

THE ASSOCIATION OF LEUKOPLAKIA WITH SYPHILIS

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Out of the 13,000 individuals examined in the "A" service of the general medical dispensary at the Episcopal Hospital since 1913 the diagnosis of syphilis has been made 500 times (about 4 per cent.); only those cases, however, with a suggestive history or symptoms were sent for a Wassermann test. The percentage of syphilis with a routine Wassermann would probably be higher. The low percentage is also due to the fact that 75 per cent. of the patients seen are women and children.

Of the 500 cases of syphilis, 295, about 60 per cent., were tertiary.

I have observed leukoplakia twenty times in the 13,000 individuals examined, and all the cases have been in subjects suffering from tertiary lues, so that leukoplakia has been present in about 7 per cent. of the tertiary cases (2 women and 18 men).

Bazin, in 1868, appears to have been the first to describe leukoplakia, which he thought was a psoriasis of the mucous membranes.

Kaposi is given credit by Emery and Chatin as first pointing out the connection between syphilis and leukoplakia, but it was not until about 1900 that this association attracted much attention. Then Fournier, after a study of more than 300 cases, stated that an antecedent syphilis was present in 90 to 95 per cent. of all cases of leukoplakia.

Since this time, however, the question as to how important a role syphilis plays as an etiological factor has been a matter of much discussion and is still to be definitely determined. The general tendency at the present day, I think, is to appreciate more and more the importance of syphilis. Many cases which without modern laboratory tests would have been classified as non-syphilitic are now shown to be associated with syphilis. Many, especially among the French writers, are emphatic in their statements that leukoplakia is always preceded by syphilis; among these are Gaucher, Landouzy and Emery.

On the other hand, many authorities, including most of the American dermatologists, seem to take the stand that while syphilis is associated in a large proportion of cases, it is not essential to the development of leukoplakia.

For instance, Stelwagon states in his 1914 edition: "The causes of leukoplakia are not clearly established. Syphilis has always been considered of etiologic import, but it occurs frequently enough in non-syphilitics to call this in question. Most of one's patients with leukoplakia, it is true, have had syphilis, but such subjects naturally suspect any manifestations and immediately seek advice, while those free from guilt in this direction are much less solicitous. Both De Bove and Schwimmer met with instances of the disease in those who subsequently acquired syphilis. It is not improbable the mercurial treatment of syphilis may, by its invoking a mild, and though often scarcely perceptible, stomatitis, be a factor of some influence. There is no doubt about the aggravating influence of smoking, and so evident is this that this practice has been considered etiologic. While its damaging, and, probably, under some circumstances, its causative action cannot be denied, yet the fact remains that the malady is also seen in those not addicted to this habit."

Schamberg states: "Perhaps the causative influence of syphilis has been exaggerated. In my experience smoking and the gouty diathesis have been the most potent etiological factors."

Others lay great stress on bad teeth, smoking, chewing, alcohol—in fact any condition which is a source of chronic irritation.

Pusey states: "When it follows syphilis it is probably the result of areas of irritable mucous membrane left in the mouth at the site of the previous syphilides."

Smoking and all other forms of chronic irritation, while they may be contributing causes, are not the main or essential factor for the development of leukoplakia. If they were, leukoplakia would be seen much more frequently.

The mouths of a large percentage of the 13,000 individuals examined were in a deplorable condition, also most of the men are inveterate users of tobacco and have used alcohol freely, so that it appears incontestable that the dominating factor in the production of leukoplakia must be something besides chronic irritation.

The view has been advanced that syphilis in some way produces a condition of the mucous membranes which favors the formation of leukoplakia, leukoplakia itself not being a true syphilitic lesion any more than an epithelioma occurring in the course of lupus is tuberculous. Holders of this view point to the ineffectiveness of antisyphilitic treatment on leukoplakia. Only two of the cases here reported have improved under the most active treatment; this seems to be the general experience. Several of the cases have had to stop or moderate their smoking owing to the tendency to ulceration. This tendency to ulcerate is more marked in cases of leukoplakia of the tongue in my experience.

That leukoplakia in a small percentage of cases does occur independently of syphilis is the view held by so many authorities of wide experience that it must be accepted as a fact for the present at least.

My own experience, however, would tend to agree with Gaucher's argument, that when an antecedent syphilis is present in 90 per cent. of the cases it probably is present in the other ten.

The importance of leukoplakia from a diagnostic standpoint cannot be too strongly emphasized. In many cases of syphilis it is the only objective symptom present to indicate the underlying disease.

Keyes states that every leukoplakia should be considered syphilitic until proved otherwise.

Leukoplakia occurs most frequently in two locations:

1. On the edges of the terminal third of the tongue.
2. On the buccal mucous membrane just back of the angle of the lips.

The distribution of the cases here reported was: Mucous membrane, both sides, 8; mucous membrane, one side, 2; tongue alone, 5; both tongue and mucous membrane, 5. In one case the lower lip was involved and in another there was a patch on the hard palate as well as on the tongue and mucous membrane.

All the men in this series of cases used tobacco.

Case I.—C. B. B., male, aged sixty-four years; married; United States; railroad fireman, April 20, 1917. Comes for dyspnea. Three sores on penis forty years ago; burnt out with caustic; lasted two weeks; no medicine taken; no secondaries. Transient paralysis of right arm, lasting one week, two years ago. Frequent frontal headaches for past two years. Wife has had four miscarriages, one stillborn and two living children.

Examination. Double aortic murmur; irregular heart action. Leukoplakia of the tongue; both angles of lips and one spot on hard palate. Wassermann plus. No change in leukoplakia following treatment. Died suddenly January 23, 1918.

Case II.—J. F., male; United States; leather worker. Wife living; no children; no miscarriages. Comes for frontal headache. Chancre of penis followed by secondary rash five years ago. Took medicine until the rash disappeared. Now complains of headaches, somnolence and tinnitus aurium. General examination negative excepting for leukoplakia at angles of the lips on both sides. Wassermann plus. No change in leukoplakia following treatment. Otherwise much improved.

Case III.—J. T. B., male, aged thirty-seven years; conductor. Comes for nervousness. Chancre of penis twelve years ago, followed by secondary rash and sore throat. Took medicine off and on for one year. One injection of salvarsan four years ago. Now complains of nervousness, headaches and indigestion. General examination negative excepting for leukoplakia of the tongue and angles of the lips, *both sides*. Wassermann plus. No change in leukoplakia following treatment. Otherwise much improved.

CASE IV.—G. H., aged thirty-five years, male; married; coppersmith. Brought to hospital for examination on account of children having hereditary syphilis. Wife living, being treated for syphilis. Four children living; two have hereditary syphilis. Denies all knowledge of infection or secondary rash. Has been treated for the past year for syphilis at another hospital.

Examination. Well nourished and developed man. Negative except for leukoplakia of lower lip. Wassermann negative.

CASE V.—M. B., male, aged thirty-six years; single; United States; bricklayer. Comes to hospital for indigestion. Sore on penis followed by sores in mouth fourteen years ago. Does not remember rash. Took medicine for two years irregularly; one injection of salvarsan six years ago. Claims to have been infected by doctor doing circumcision. General examination negative excepting for leukoplakia left side of tongue. Wassermann negative.

CASE VI.—J. W., male, aged forty-five years; United States; teamster. Comes to hospital for sore mouth. Wife has tertiary syphilis; has had one miscarriage, no children. Chancre of penis twenty-five years ago, also bubo and sores in mouth at this time. Does not remember rash. Took pills off and on for two years. Shooting pains in legs for two years. Diplopia, transient, for past year. Attacks of vertigo for past year.

Examination. Typical case of tabes. Marked leukoplakia at internal angle of lips, both sides, part of which is ulcerated; also chronic glossitis of tongue. Marked improvement of tabetic symptoms and healing of ulcerations of mouth following treatment. Wassermann plus.

CASE VII.—S. W., male, aged forty-five years; United States; watchman. Wife living, neurotic. Four children living. Several miscarriages. Chancre followed by secondary rash twenty-four years ago. Took medicine for two years. Attacks of vertigo for past year. Brought to hospital unconscious, with paralysis of left side. Was well again in one week.

Examination. Gait slightly spastic on left side; reflexes increased on this side. Large scar on right side of tongue, probably due to gumma. Leukoplakia of tongue. Wassermann plus. Marked improvement, no change in leukoplakia, which ulcerates whenever he attempts to smoke.

Case VIII.—F. T., male, aged sixty-eight years; United States; stevedore. Comes to hospital for "stomach trouble." Wife died of cancer of the breast. Always healthy until present trouble, except for injuries due to his work. Fracture of skull six years ago. Denies any knowledge of syphilis. Stomach trouble for the past three years; vomits at times; now being treated for lacerations of the left arm and keloid scar.

Examination. Negative except for arm and old trephine scar. Leukoplakia of tongue and angle of lips on both sides. Some improvement of leukoplakia after several injections of salvarsan. Wassermann plus.

Case IX.—S. K., male, aged sixty years; United States; leather finisher. Wife died of pulmonary tuberculosis. No children. One miscarriage. Comes to hospital on account of trouble when walking in dark. Denies syphilis. Gonorrhea seven times. Diplopia eight months ago, lasted two months. Shooting pains for two years in legs.

Examination. Typical case of tabes. Chronic glossitis of tongue. Leukoplakia at angle of lips of both sides. Wassermann plus. No change in leukoplakia following treatment.

Case X.—R. McG., male, aged thirty-three years; United States; married; weaver. Comes to hospital for pain in the chest. Wife living; six children living, one died suddenly. Chancre of penis fifteen years ago, followed by secondary rash. Took medicine off and on for two years, also at odd times since on account of sore mouth.

General Examination. Separation of cartilages from three lower ribs on left side, due to accident. Chronic glossitis, with leukoplakia on tongue. Wassermann plus. Leukoplakia improves when he stops smoking.

Case XI.—G. N., female, aged thirty-five years; married; United States. Comes for loss of voice. Ten miscarriages, self-induced. Sore throat, followed by general eruption, seven years ago; followed some weeks later by iritis of left eye. Denies knowledge of primary sore. Has taken treatment irregularly since.

Examination. Acute laryngitis. Thyroid decidedly enlarged; many small brownish scars on arms and elbows dating from eruption. Leukoplakia at angles of mouth on both sides. Patient denies smoking. Wassermann plus.

Case XII.—W. B. J., male, aged forty-one years; United States; machinist. Comes to hospital for blood examination. Wife living. No children. No miscarriages. Sore on penis thirteen years ago. Denies secondaries. Took medicine (pills) for eighteen months. Nervous breakdown four years ago. Two years ago developed sore at right angle of mouth, involving skin and mucous membrane. Has taken mixed treatment off and on since.

Examination. Negative except for marked leukoplakia inside right angle of lips. Wassermann plus.

Case XIII.—W. W., male, aged twenty-nine years; United States. Comes to hospital for nervousness. Wife and four children living and well. Three sores on penis thirteen years ago; no secondary rash but was in bed several weeks "with weakness." Another sore on penis one year ago, which lasted one week. No other objective symptoms, but has been nervous, with numbness of legs since.

Examination. Many dark scars on shins which patient claims are due to boils. Leukoplakia at angles of lips on both sides. Wassermann plus.

Case XIV.—T. Q., male, aged sixty-six years, married; United States. Comes to hospital for sore on right wrist. Wife living; one miscarriage; five children living. Smallpox in 1872. Chancre of penis followed by rash twenty years ago, took medicine for six weeks.

Examination. Ulcer on the right forearm just above the wrist on the flexor surface, the size of a quarter dollar, suppurating. Sugar in urine. Leukoplakia at angles of the lips, on both sides; ulcerated on left side. Wassermann plus. Leukoplakia improved after treatment and diet.

Case XV.—M. M., female, aged fifty-six years; married; Ireland; housewife. Comes to hospital for headaches. Married twice. Divorced from first husband. Two children living and one miscarriage. Menopause ten years ago. Frontal headache frequent for past four years. Tongue sore off and on for four years. Eczema of palms of hands and soles of feet for past two years. Has been treated constantly for two years, with no improvement.

Examination.—Chronic eczema of hands and feet; atrophy of mucous membrane of tongue; several large fissures; leukoplakia covering sides of tongue. Wassermann negative. Headache and eczema cured with mixed treatment. No change in leukoplakia.

Case XVI.—J. C., male, aged thirty-six years; married; United States; water-filter attendant. Comes to hospital for sore tongue. Wife living; no children; no miscarriages. Chancre of penis followed by rash five years ago. Has taken medicine irregularly since.

Examination. Ulcerating gumma of tongue; leukoplakia of tongue and angles of lips on both sides. Wassermann plus.

Case XVII.—E. H., male, aged sixty-eight years; married; United States; janitor. Comes to hospital for pain in abdomen after eating. Chancre and secondary rash when eighteen years old. Does not remember how long he took treatment, for some months at least. Gonorrhea at twenty years. Wife living; one miscarriage; one daughter.

Examination. Gall-bladder palpable and tender. Refused operation. Marked leukoplakia of tongue; anterior part ulcerated and subsequently part of tongue was excised. Seen one year later; no recurrence. Wassermann negative.

CASE XVIII.—P. H., male, aged forty-four years; married; Ireland; laborer. Comes to hospital for rash on back. Wife died of typhoid fever. Three children living. Chancre on penis followed by rash sixteen years ago; treated for six months. Was in bed for three months with pains in joints a year later; joints not swollen, only painful. Pains off and on since then.

Examination. Pupils unequal; right larger; react. Dr. Hopkin reports specific choroiditis. Leukoplakia at angles of lips on both sides. Dermatitis of back due to belladonna plaster. Wassermann negative. Much improvement under treatment; no change in leukoplakia.

CASE XIX.—P. M., male, aged fifty years; single; motorman. Comes to hospital for cold. Gonorrhea thirty years ago. Chancre of penis and general rash treated at Episcopal Hospital eight years ago. Taking treatment off and on ever since.

Examination. Negative except for leukoplakia at angles of lips on both sides. Wassermann negative.

CASE XX.—W. K., male, aged fifty-seven years; widower; United States; watchman. Comes for headache. Wife died, cause unknown. No children. Denies syphilis. Headache constantly for two years.

Examination. Almost total deafness; bone conduction *nil.* Left leg artificial from above knee (accident). Eyes: View of fundus not obtainable due to cataract. Marked leukoplakia of tongue and angle of lips. Wassermann negative. Headaches improved under mixed treatment. Developed insanity and now in the Philadelphia General Hospital.

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REPORT OF THE DENTAL AND ORAL SURGICAL DEPARTMENT

By JAMES R. CAMERON, D.D.S.
VISITING DENTAL SURGEON TO THE HOSPITAL

AND

MAURICE C. BOND, D.D.S.
RESIDENT DENTAL SURGEON IN THE HOSPITAL

This report will be more of a tabulation of the work done during the past twelve months rather than a recitation of case reports. For the greater part of the year the department was minus a chief, who was in the military service, and also for a short time was without a resident, owing to the same cause. Now that the work can be carried on as formerly we hope to accomplish much in the future. The war record of the dental profession has more than demonstrated the need and advisability of having a dental service in all large and well-conducted hospitals. Possibly within the near future we may be successful in having added to the present dental equipment one in close proximity to the medical and surgical wards, thereby permitting a large number of ward cases to receive dental attention, especially prophylactic treatments. The majority of patients on the wards are usually indifferent to the value of a clean mouth. As the mouth is the gateway to the human body it is not unreasonable to suppose that a healthy mouth would aid materially in shortening the stay of many patients in the hospital. Today, when we know so much about oral foci and their relationship to systemic disease, and there still is so much more to learn about this subject, the oral care of patients for both medical and surgical conditions is quite an important phase of the treatment.

During the course of training required for graduation from the training school for nurses a short period of that time devoted to the study of oral hygiene and its application in hospital treatment would be, in the writer's opinion, an excellent thing for the welfare of patients. Whenever possible patients about to undergo surgical operations would undoubtedly be better prepared for such an ordeal when the mouth had previously been freed of calcareous deposits, loose roots, etc.; especially is this so in children whose tonsils are to be removed.

The following is the tabulated report of the work done in the dental clinic of this hospital and in A. E. F. Base Hospital No. 34:

Number of patients treated	921
Extraction of teeth	1362
Permanent fillings	56
Cement fillings	124
Root canals treated	306
Ward patients treated	130
Necrosis of maxillæ	6
Fractures of maxillæ	20
Impacted teeth	11
Alveolar abscess (acute)	10
Submaxillary abscess	2
Empyema maxillary sinus	2
Malignancy maxillary sinus	1
Epulis (maxilla)	2
Gas-oxygen anesthesia	225
Procaine conduction anesthesia	70

REPORT OF THE DENTAL DEPARTMENT OF BASE HOSPITAL NO. 34, A. E. F., by GEORGE A. COLEMAN, D.D.S.,

Defective fillings	47
Dental caries	2769
Dento-alveolar abscess	662
Devitalized pulp	307
Erosion	1
Erupting teeth (painful)	8
Empyema maxillary sinus	4
Fractured jaws	67
Fractured teeth	6
Gingivitis (simple)	4
Impacted teeth	35
Pulpitis, acute	213
Pulpitis, chronic	309
Salivary deposits	460
Vincent's angina	239
Loose inlays	8
Loose crowns	29
Loose bridges	12
Hyperemia	88
Amalgam fillings	1409
Amalgam oxyphosphate	469
Gold inlays	11
Oxyphosphate cement	352
Root canal operations	241
Synthetic porcelain	497
Gold shell crowns	5
Gold and porcelain crowns	1
Porcelain crowns	2
Crowns reset	22
Bridges reset	12
Inlays reset	8
Partial dentures	96
Partial lower dentures	66
Swaged upper metal splints	20
Swaged lower metal splints	47
Teeth extracted	1045
Alveolar abscesses lanced	80
Pulps devitalized	56
Pulps extirpated	152
Pulps capped	13
Calculus removed	460
Prophylaxis	212
Roots amputated	22
Teeth treated	850

TWO CASES OF COMPOUND FRACTURE OF THE JAW

By CARL C. GAGE, D.D.S.
FORMERLY DENTAL RESIDENT IN THE HOSPITAL

Case I.—Case report: Compound comminuted fracture of maxilla, complicated by facial lacerations. Patient admitted April 15, 1918. Katie F., Austrian, aged forty-eight years.

Condition on Admission. Patient struck by broken belt in Pennsylvania Railroad shop; is bleeding from mouth and nose with several bleeding lacerations of the face. One large cut below the chin extending from the mental foramen on right side to that on the left, and exposing the lingual surface of the mandible.

Face badly swollen especially in the region of the eyes. Intraoral examination reveals that the upper incisor teeth are broken loose in the alveolar process en masse. Palatal suture is open and the maxilla can be moved laterally revealing fracture below malar bone bilateral. Mechanical abrasions of the teeth very marked. Radiograph confirms clinical diagnosis.

Treatment consisted in cleansing and suturing the facial lacerations. Barton bandage applied and ice compresses used to reduce the swelling. Local measures used failed to control hemorrhage of nasal wound and 10 c.c. of horse serum was given about nine hours after admission. This apparently checked the hemorrhage.

For the following forty-eight hours ordinary antiseptic measures were used to keep the mouth clean. By this time the swelling had been reduced sufficiently to permit impressions of both jaws being obtained and interdental splints were constructed. These splints consisted of a full upper and lower nickel silver splint with extraoral extension. The upper was not soldered to the lower but after application to the teeth the two splints were later fixed to each other by wires attached to hooks. The fracture was reduced and brought into correct alignment, the splints cemented into place and several hours later after the cement had hardened sufficiently the upper was secured to the lower splint by means of the wires mentioned above. The extra-oral extensions were used for upward and outward pressure by the use of a bandage passed through a loop.

Patient was comfortable for three or four days when her temperature rose to 102° F. Radiographic examination revealed slight cloudiness of the frontal and maxillary sinuses. Her condition at this time was such as to cause suspicions of a beginning meningitis. This condition however cleared up within a few days. The mouth and external wounds were cleansed daily with Dakin 2 per cent. solution in oil. The patient made an excellent recovery and the splints were removed in about four weeks, firm union having been obtained.

Discharged May 11, 1918.

Case II.—Compound comminuted fracture of the maxillæ, involving nasal floor and compound comminuted fracture of the mandible.

Patient admitted April 22, 1918, Marion F., male, Italian, aged forty-six years.

Condition on Admission. Patient states that he fell from a scaffolding thirty feet above the ground. He presents a diffuse ecchymosis over the right eye and laceration of the lower lip severing the vermilion border. Intra-oral examination shows a most filthy mouth, with the lower incisor teeth and alveolar process movable *en masse.* In the left side the mandible is fractured in the region of the first molar tooth and involving the roots of this tooth.

Treatment consisted in cleansing the external wound and suturing the same. Fractures reduced and held in alignment by the aid of a tight Barton bandage. Ice compresses applied for the purpose of reducing the swelling. Within forty-eight hours the mouth was given a thorough prophylactic treatment. The lower left first molar was now extracted. A 2 per cent. Dakin oil spray was used in the mouth and nose every three hours and a mouthwash of potassium permanganate, 1 to 1000, every two hours. The following day wire ligatures were placed around several upper and lower teeth, on either side of the fracture and the jaws brought into correct apposition and secured one to the other. These wires were retained for about four weeks, during which time the patient received nourishment through a tube. On removal of the wires firm union was found to have taken place.

Discharged May 25, 1918.

I desire to express my indebtedness to Dr. H. C. Deaver for the privilege of reporting these cases, which were treated in his service.

TONSILLECTOMY UNDER LOCAL ANESTHESIA

By EDWIN P. LONGAKER, M.D.
ASSISTANT IN THE AURAL AND LARYNGEAL CLINIC OF THE HOSPITAL

Following one's experience in the army, radical changes are often made. During the summer of 1918 many soldiers were sent for operation to a base hospital where the writer was located. Before deciding on a tonsillectomy one of three or four things was sought: (1) Pus in the tonsil, found by pressure on the tonsil, often undisclosed unless this be done; (2) streptococci in the throat (95 per cent. disappeared a few days after operation); (3) some external condition, *i. e.*, "rheumatism," where the tonsil was under suspicion, as a focus of infection; (4) unusually large tonsils or cases in which the history was one of frequent attacks of tonsillitis or quinsy.

It soon became evident that as there were so many cases a general anesthetic was out of the question.

Cocain had been used in a few scattered cases prior to the rush of large numbers. The results were unsatisfactory; two cases nearly ended disastrously in which $\frac{1}{8}$ of 1 per cent. solution was thought to have been used.

The combination found to work the best was novocain, $\frac{1}{2}$ per cent. with adrenalin, $\frac{1}{32,000}$. All instruments were thoroughly boiled between operations, and gowns were changed after each case. Twenty to thirty operations were done almost daily. Prior to the injection of novocain and adrenalin the pharynx, posterior tongue and tonsils with peritonsillar tissue were touched with 4 per cent. cocain.

A dental needle with a short point (less than $\frac{1}{4}$ inch), and with a heavy shoulder, was used for injecting. The heavy shoulder allowed only a small puncture. This needle was found to be ideal.

After 100 cases had been done there appeared in 4 or 5 per cent. of cases nasty abscesses in the peritonsillar tissue three to five days after operating, which had to be incised just as any peritonsillar abscess. The technic was gone over carefully, and to make doubly sure, the points of injecting the novocain were touched with iodin prior to the injection; and, following the operation, the fossa was swabbed with tincture of iodin. The percentage of infections continued until strict care was taken to keep out of the tonsil when making the injection, only injecting into the pillars and behind the tonsil.

INSTRUMENTS USED. Other than the needle mentioned these were employed: A long metal syringe, a long tonsil grasp, Dean's straight and angular tonsil knives and a Tyding snare, in addition to several hemostats. A long tonsil grasp does away with the necessity of a tongue depresser, the grasp acting as one. The Dean tonsil knives have very sharp edges with a blunt end, allowing a clean cut down to the capsule, and the blunt end pushes the tissues away from the tonsil. Tyding's snare has the pistol-grip and allows a very quick snap and separation, something to be desired in a local tonsillectomy.

In about 500 cases done during "service" 2 per cent. bled, requiring attention; only 1 case needed suturing, the others were stopped by hemostats allowed to remain on three or four minutes. In 60 cases done in the clinic of the Protestant Episcopal Hospital and in private practice since then, 1 required hemostatic pressure. None of the 30 patients had any postoperative infection.

Rarely was an adenoid removed under local anesthesia, and when done the operation was rather unsatisfactory.

Local anesthesia should be painless, or so nearly so that it does not annoy the patient; if painful the operator is at fault.

The adrenalin helps to give hemostasis, allowing a dry field during operation, and does not predispose to later bleeding. Without a dry field the tonsil may not be entirely removed. I believe 2 per cent. of adults will bleed whether done under local anesthesia or ether; this does not include children.

Nervous individuals may, with good results, be given by hypodermic one hour prior to operation, morph. sulph., gr. $\frac{1}{4}$ or $\frac{1}{6}$, combined either with atropin, gr. $\frac{1}{100}$ or scopolamin, gr. $\frac{1}{100}$.

Many of our patients should not take ether. The writer recently operated on a woman, aged sixty years, who had marked arterial hypertension and chronic interstitial nephritis, with severe rheumatism. Pus was in both tonsils. She stood the local anesthetic well.

Another patient, a man, aged thirty-five years, with pus in one tonsil and a blood-pressure of 170 to 110, had a local tonsillectomy in July, 1919, and in one month the pressure was 135 to 100, and has remained at that level since then (seven months ago). Children are not subjects for local anesthesia. Adenoids cannot be anesthetized properly.

Postoperative infections are due to forcing infected material through the tonsil into the peritonsillar tissue; strict care should be taken with technic. Local anesthesia has a distinct place in tonsillectomy.

A PRACTICAL METHOD OF ISOLATING ACUTE PULMONARY INFECTION CASES IN A HOSPITAL WARD

By WILLIAM S. CUNNINGHAM, M.D.
RESIDENT PHYSICIAN IN THE HOSPITAL

During the recent influenza epidemic in Philadelphia the medical wards of the hospital were unavoidably crowded, so it became necessary to put the patients' beds close together, two and even, in some instances, four beds being placed in the space normally occupied by a single bed. Nearly all these patients had violent attacks of coughing, with profuse expectoration; hence the problem of reinfection of a patient immediately arose: *e. g.*, a convalescing type ii pneumonia might be unavoidably occupying a position adjacent to a type iii or an acute influenza case.

Several plans were tried early in the congested period in order to prevent transmission by droplet infection, such as alternating the heads of the beds and putting the ordinary screens around the heads of the beds, but these proved very unsatisfactory for many reasons. Other methods were contemplated but deemed impractical. During the stage of experimentation three separate cases of reinfection in convalescing pneumonias occurred which proved fatal in one patient, who probably acquired it from his neighbor, as the laboratory tests indicated.

The accompanying illustrations give some idea of the very excellent plan adopted in order to isolate each individual patient as much as possible. The structure is simple enough: it is easily kept in position, the single draped sheet is easily changed for a clean one, it does not alter the tidiness of the ward, and still more important it makes it possible for a small staff of nurses to watch a number of delirious patients. It allows the patients to be moved to the porch with comparative ease and there also have the same degree of protection in a crowded area.

It is very interesting to note that after these screens had been installed no cases of reinfection were traceable to transmission from a neighboring patient.

The plan of the screen is as follows: Upright beams are attached at the middle of the head and foot of the ordinary hospital bed. About four feet from the plane of the bed are cross arms extending the width of the latter. Both ends of these arms are connected by wires, and over the head part is draped a single bed sheet. On the right side the sheet is made to extend down to about two feet from the foot and on the left side about one foot from the head. So when the beds are placed next to one another there is a sheet partition between each. At the left side is placed the table holding the sick-bed accessories.

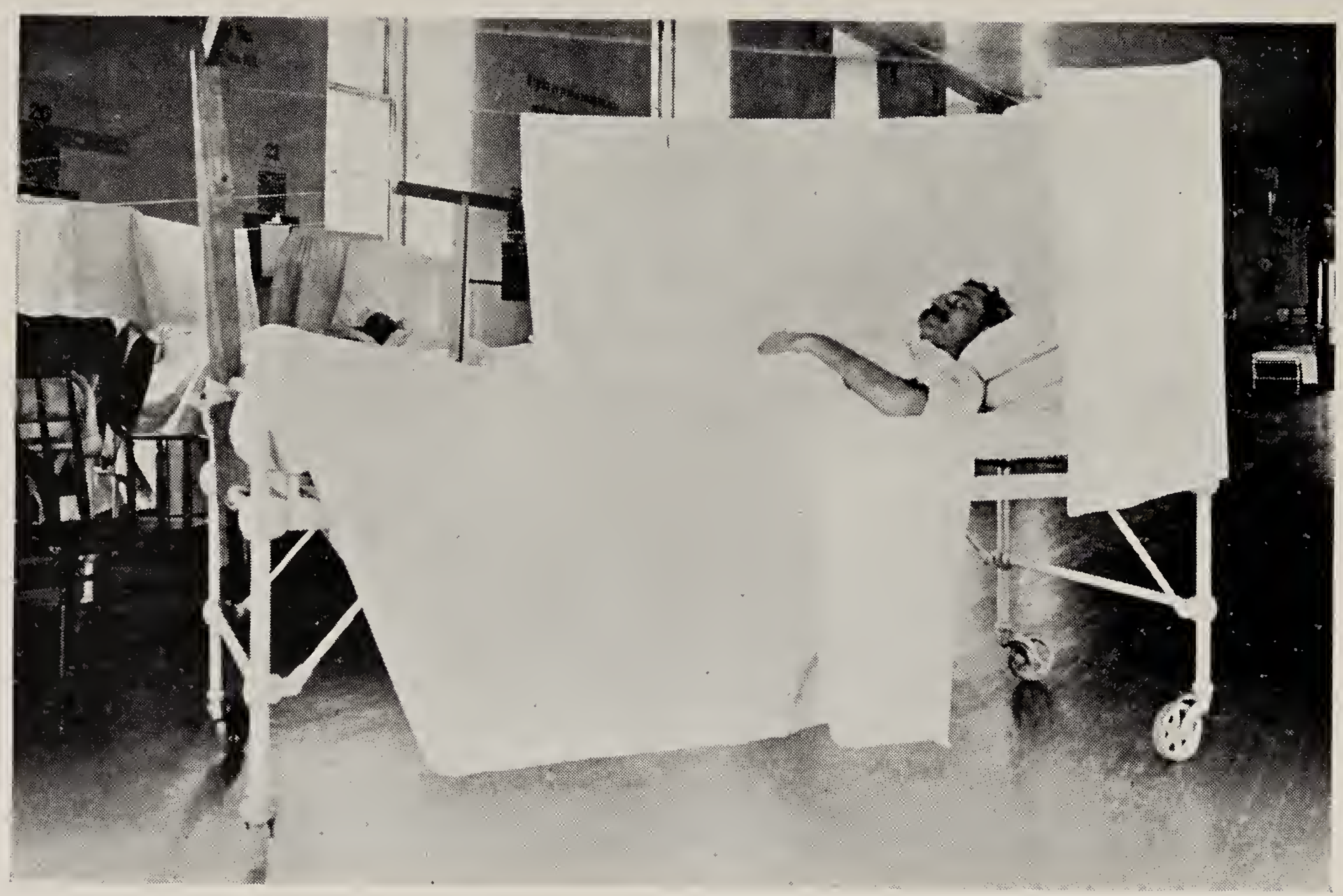

FIG. 107.—Isolation by sheets: the sheets are shown in position.

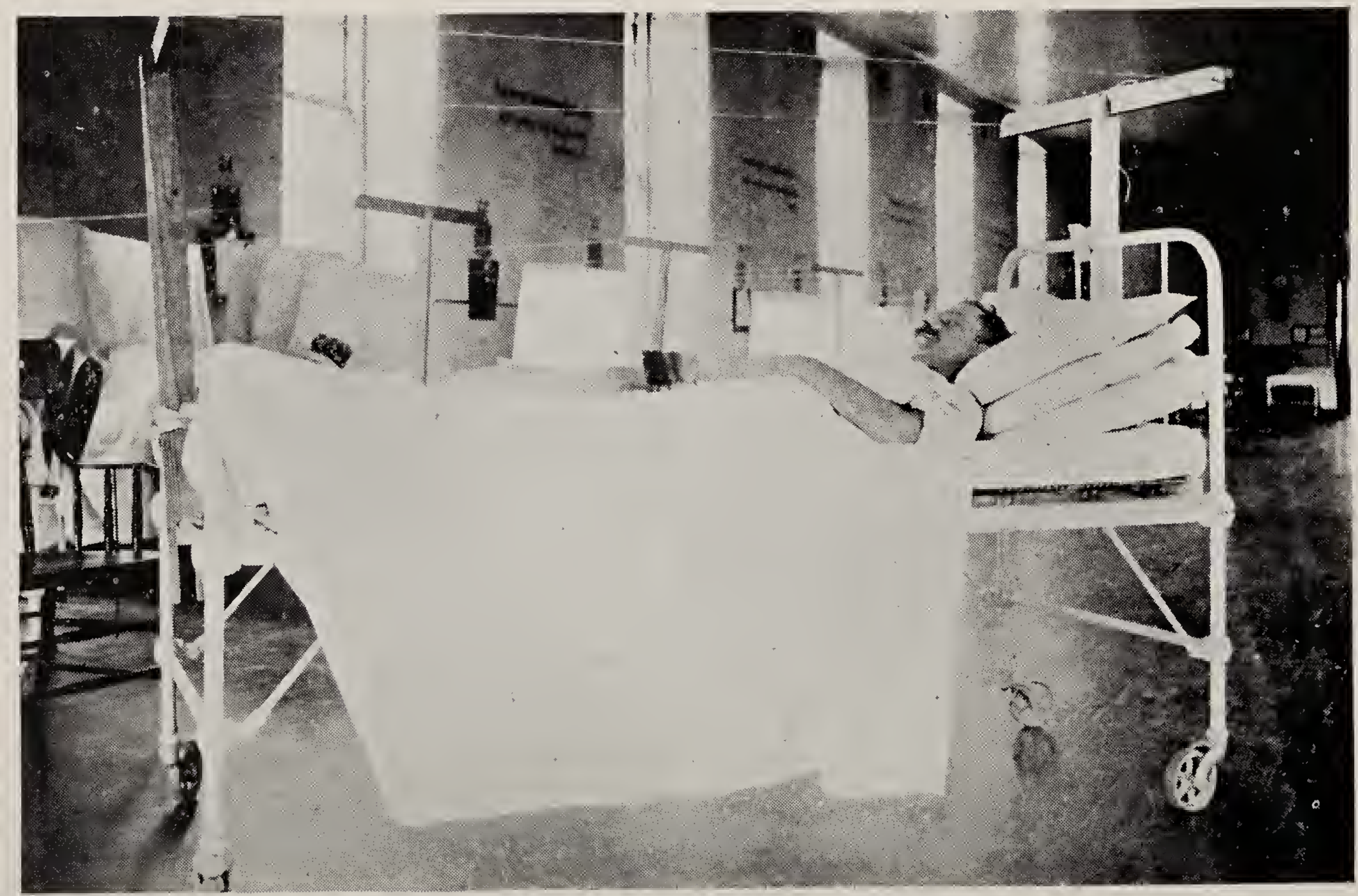

FIG. 108.—Isolation by sheets: showing the simple T-frames and cords on which the sheets are hung.

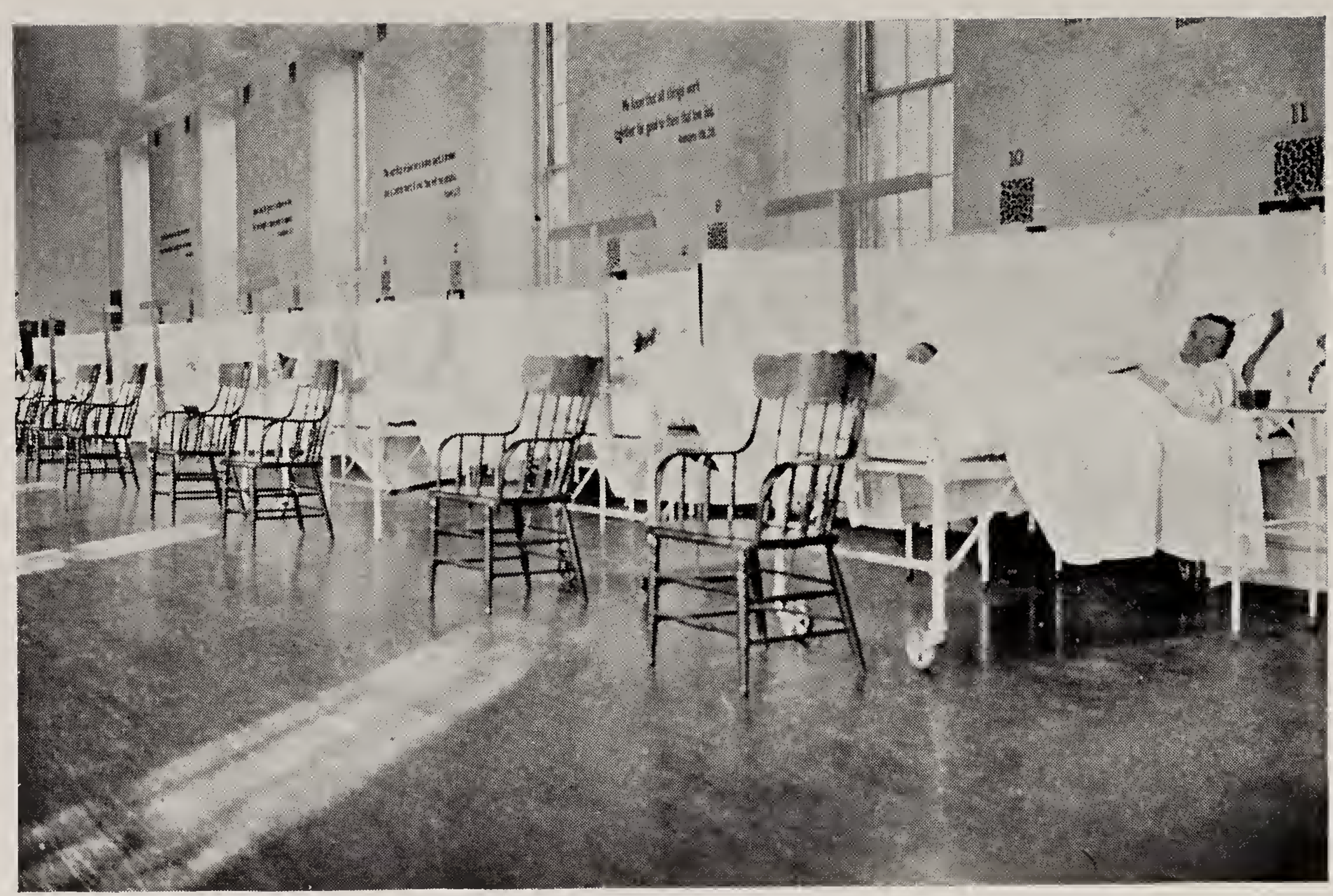

FIG. 109—Isolation by sheets: general appearance of the ward, with the beds all open on the left of each patient, screened on the right.

ADVANCED CARCINOMA OF THE BREAST: NO RECURRENCE SEVEN YEARS AFTER OPERATION[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

Dr. Astley P. C. Ashhurst presented this patient as an encouragement toward doing thorough and widespread excisions for carcinoma. If in such a case as the present cure can be obtained by such means, how much more certainly should cure be anticipated if the same type of radical operation were uniformly adopted in early cases? He added that he had had occasion recently to operate on a case of widespread local recurrence in a case of carcinoma of the breast one year after another surgeon had done a very incomplete operation, although the clinical diagnosis had been carcinoma from the first. This fact seemed to indicate that some surgeons were still so skeptical of cure ever being attained that they considered it not worth their while to expend the time and skill necessary for a complete extirpation. In the case of recurrence, to which reference had just been made, the surgeon at the first operation not only did not excise the pectoral muscles but he did not expose the axilla at all, nor did he even remove all the glandular tissue of the breast; yet the clinical diagnosis, confirmed by histological study, had been carcinoma from the first! He desired also to make a plea for habitual roentgenization of carcinoma patients after operation. In the case of the patient now presented, postoperative roentgen therapy was continued a long time.

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[1] Presented at a meeting of the Philadelphia Academy of Surgery, October 6, 1919. Reprinted from Annals of Surgery, 1920, i, 94.

The patient, a woman, aged forty-five years, was admitted to the Episcopal Hospital August 3, 1912. There was a typical "rose ulcer," 7.5 by 5 cm. in diameter, in the upper outer quadrant of the right breast. This ulcer had commenced six months previously, and for two years before that time the woman had known she had a tumor in the breast. When examined there was a hard tumor, the size of a goose egg, beneath the ulcer. This tumor was freely movable in all directions. There was a palpable and visible mass of lymphatics in the axilla, which also was movable. The ulcer was covered with an adherent gray slough. No lymph nodes were palpable above the clavicle. There was tenderness over the liver, but no evidence of metastasis to this organ. The left breast was normal.

Operation, under ether anesthesia, was done August 5, 1912, including a clean dissection of the entire axilla, and removal of its contents, both pectoral muscles, and a wide area of superficial and deep fascia (beyond the midline at the sternum and down almost to the umbilicus) in one mass. It was possible to close all the wound except for a space 5 by 2.5 cm. below the clavicle. The time of the operation was three and a half hours, and at its conclusion the patient received one liter of saline solution intravenously, being almost pulseless. (The excised specimen is illustrated in Plates XII and XIII of the speaker's *Text-book of Surgery.*)

By the second day after operation convalescence was established. Many of the sutures did not hold, some of the skin edges became necrotic, and six weeks after operation there was a clean granulating area the size of two palms. On September 30 this had contracted to an area the size of one palm, and this was covered with Thiersch grafts. Meanwhile roentgen-ray treatment was pursued for a number of months.

The patient was kept under observation:

March, 1914. Her health was feeble and she could not do much work. There was still an unhealed area, 2 cm. in diameter, which scabbed over from time to time. There was no swelling of the hand or the forearm, and only slight edema of the upper arm; but the edema increased if the arm was used much. She could get her hand to her head and back to her buttocks, but both motions were weak and the latter painful. The extensive cicatrix sometimes caused a sticking pain in her lung, but there was no evidence of pulmonary metastasis.

August, 1915. Condition about the same.

September 24, 1919. Since the last note the patient's general condition has markedly improved. She does her own housework and looks after an epileptic son. The entire right anterior thorax is covered by skin tightly adherent to the ribs, but there is no evidence of recurrence locally or of metastasis. She has good use of her arm, raising her hand easily above her head, and putting it without difficulty to the small of her back. Except for slight weakness she thinks it as useful as her left arm. There is scarcely appreciable swelling of the hand and none of the forearm or arm. During the past year her appendix was removed at the Philadelphia General Hospital.

Dr. Ashhurst, in closing the discussion of this case, said that he had no doubt that the malignancy of different specimens of cancer varies a great deal. He thought also that diminished lymphatic activity is to be taken into consideration. In patients nearly eighty years old it seems useless to do an extensive operation. If we merely amputate the breast there is not likely to be subsequent trouble. But it ought to be remembered how far the mammary gland extends beyond that which one sees. If one thinks the glandular tissue occupies a very small area he shall be deceived: it extends nearly up to the clavicle, out into the axilla and down toward the epigastrium. The entire mammary gland should be removed even in these incomplete operations on very aged patients.

A COMPARATIVE STUDY OF 105 CASES OF PNEUMONIA THAT CAME TO AUTOPSY IN HOSPITAL CENTER, NANTES, FRANCE (1918)[1]

By JOHN WALKER MOORE, M.D.
LOUISVILLE, KY.

BENJAMIN F. BUZBY, M.D.
PHILADELPHIA, PA.

AND

JOHN DAVIS PAUL, M.D.
PHILADELPHIA, PA.
FORMERLY RESIDENT PHYSICIANS IN THE HOSPITAL

After a general study of the cases of pneumonia that came to autopsy in our center, we adopted a gross classification, which we feel is not entirely satisfactory, but apparently meets the needs of the material at hand as no other gross classification would do.

The classification is as follows:

Patchy bronchopneumonia.
Massive bronchopneumonia.
Lobar pneumonia.

The description of these lesions is embraced under the heading of gross pathology.

For a comparative study of 105 cases of patchy bronchopneumonia, massive bronchopneumonia and lobar pneumonia, we have chosen the following headings:

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[1] Eighty-five per cent. of the cases was autopsied in Laboratory of Base Hospital No. 34.

IOLOGY	Hemolytic streptococcus.										Non-hemolytic streptococcus.										Streptococcus type undetermined.										Non-hemolytic streptococcus and Bacillus influenzæ.										Non-hemolytic streptococcus and pneumococcus.									
	Broncho.								Lobar.		Broncho.								Lobar.		Broncho.								Lobar.		Broncho.								Lobar.		Broncho.								Lobar.	
	Patch.				Mass.						Patch.				Mass.						Patch.				Mass.						Patch.				Mass.						Patch.				Mass.					
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IOLOGY	Pneumococcus Type I										Pneumococcus Type II										Pneumococcus Type III										Pneumococcus Type IV										Pneumococcus Type undetermined.									
	Broncho.								Lobar.		Broncho.								Lobar.		Broncho.								Lobar.		Broncho.								Lobar.		Broncho.								Lobar.	
	Patch.				Mass.						Patch.				Mass.						Patch.				Mass.						Patch.				Mass.						Patch.				Mass.					
	S	B	A	I	S	B	A	I	D	B	S	B	A	I	S	B	A	I	D	B	S	B	A	I	S	B	A	I	D	B	S	B	A	I	S	B	A	I	D	B	S	B	A	I	S	B	A	I	D	B
e)						2														I										3											4									I
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IOLOGY	Pneumococcus and gas.										Pneumococcus and Bacillus influenzæ.										Bacillus influenzæ.										Meningococcus, pneumococcus and Bacillus influenzæ.										Menin strep			
	Broncho.								Lobar.		Broncho.								Lobar.		Broncho.								Lobar.		Broncho.								Lobar.		Bron			
	Patch.				Mass.						Patch.				Mass.						Patch.				Mass.						Patch.				Mass.						Patch.			
	S	B	A	I	S	B	A	I	D	B	S	B	A	I	S	B	A	I	D	B	S	B	A	I	S	B	A	I	D	B	S	B	A	I	S	B	A	I	D	B	S	B	A	I
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The chart shows the frequency with which the different types of organisms are associated with the different pathological lesions, together with the anatomical portions of lung involved. The letters S, B, A, I and D located at top of small columns, signify serum, blood, abscess, infarct and dry.

Note (1) the absence of lobar pneumonia and the prevalence of massive bronchopneumonia with the Streptococcic group and with organisms associated with the influenza bacillus, and (2) the frequency with which lobar pneumonia is associated with the different types of the pneumococcic group.

I. Relative Proportion of Cases and the Months in which They Occurred:

Of the 29 cases of patchy bronchopneumonia, 3 per cent. occurred in June, 18 per cent. occurred in August, 6 per cent. occurred in September, 20 per cent. occurred in October, 35 per cent. occurred in November, 18 per cent. occurred in December.

Of the 42 cases of massive bronchopneumonia, 7.2 per cent. occurred in August, 2.3 per cent. occurred in September, 52.3 per cent. occurred in October, 21.5 per cent. occurred in November, 16.7 per cent. occurred in December.

Of the 34 cases of lobar pneumonia 6 per cent. occurred in July, 17 per cent. occurred in August, 41 per cent. occurred in October, 24 per cent. occurred in November, 12 per cent. occurred in December.

II. Relative Duration of Diseases:

Patchy bronchopneumonia average 8.2 days until death.
Massive bronchopneumonia averaged 6.5 days until death.
Lobar pneumonia averaged 6.7 days until death.

III. Clinical Course of Disease. (*a*) Physical signs. The number of histories of any value available in this series was 19 in 29 cases of patchy bronchopneumonia, 28 in 42 cases of massive bronchopneumonia and 28 in 34 cases of lobar pneumonia. Even in these histories, in most cases the data which might be of value in distinguishing one pathologic type from another were decidedly incomplete. In the massive type of bronchopneumonia the onset by chilliness, cough and pain in the back, limbs, head and chest was more constant than in the other two types. Diarrhea marked the onset in 25 per cent. of the massive cases, whereas in only 1 case of patchy bronchopneumonia and in no cases of lobar was it present at the onset.

In the secondary symptoms the percentage of cases showing delirium was comparatively higher in the patchy type (44.5 per cent.) than in the massive (28.5 per cent.) and the lobar type (25 per cent.). Cyanosis, however, was much more frequently encountered in the massive type (75 per cent.) than in the patchy (26.3 per cent.) and the lobar (50 per cent.) types. Further than this the symptoms of onset and secondary symptoms show nothing of interest or importance.

(*b*) Temperature, pulse and respiration. Of the 105 cases of pneumonia that came to autopsy only 55 histories contained sufficient data for studying temperature, pulse and respiration. Of this series 12 were patchy bronchopneumonia, 22 massive bronchopneumonia and 21 lobar pneumonia. In comparing the mean temperature, pulse and respiration of the three types of pneumonia we find the following characteristics:

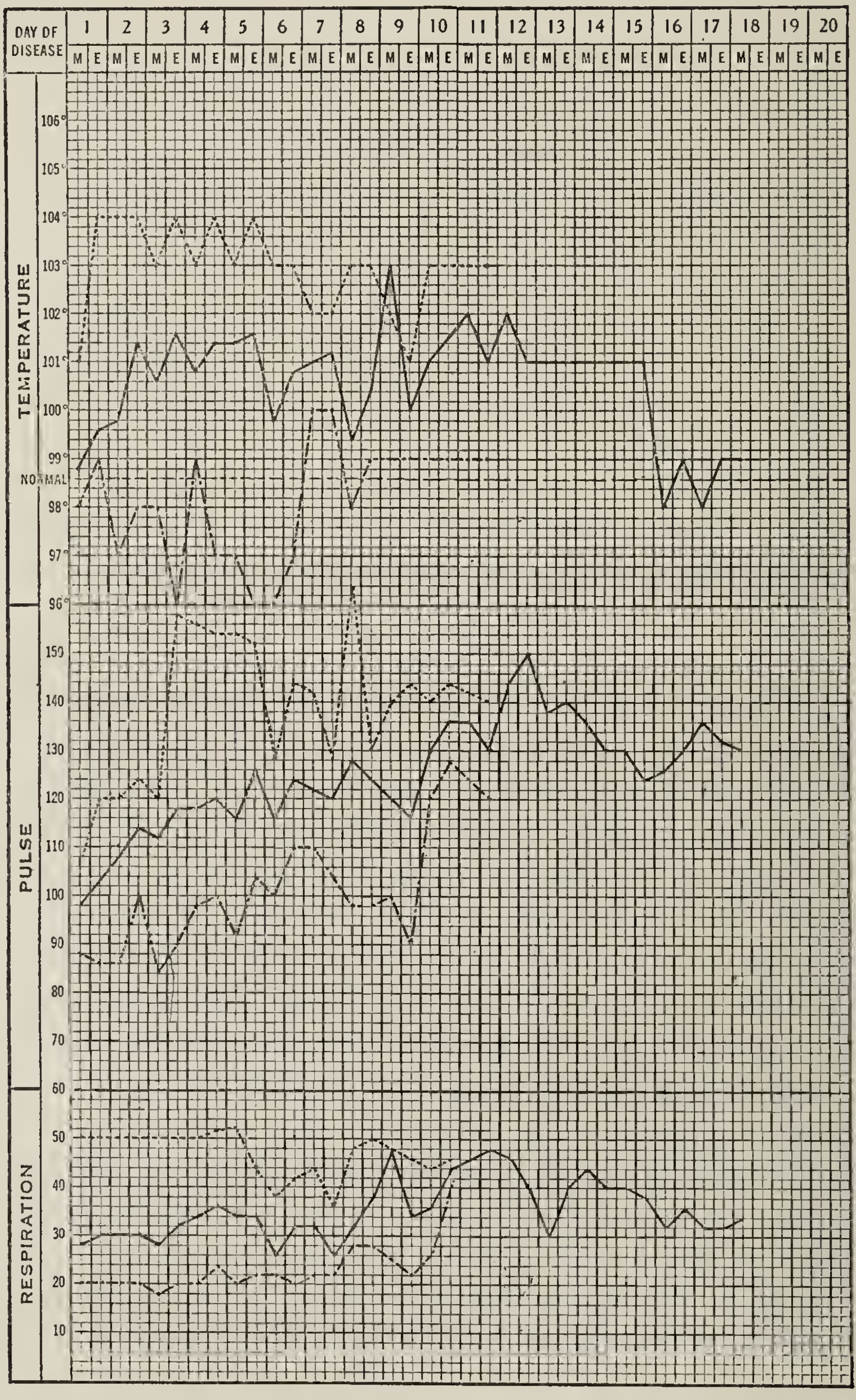

Fig. 110.—Patchy bronchopneumonia (Composite chart), showing maximum, medium and minimum range of temperature, pulse and respiration.

In patchy bronchopneumonia the temperature in the first twenty-four hours began about 99°, rising rapidly until the evening of the second day, at which time the temperature reached 101.6° and continued about this point until the evening of the fifth day;

in massive bronchopneumonia the temperature in the first twenty-four hours ranged between 101° and 102.6°; gradually rose to 103.8° on the morning of the third day; dropping on the morning of the fourth day to 101°; jumping on the evening of that day to 102.8° and remaining there until the evening of the fifth day; in lobar pneumonia in the first twenty-four hours the temperature ranged between 102.6° and 103.8°, and it continued to fluctuate between 102.8° and 103.4° until the evening of the fifth day. It is seen, therefore, in patchy bronchopneumonia the temperature starts at a low degree, jumps rapidly, though never attaining a degree over 101.8, and has a tendency to remain at the maximum temperature; whereas in massive bronchopneumonia the temperature begins at a higher degree (101°), attains its height in the first twenty-four hours and shows a tendency to fall; and lobar pneumonia starts at a still higher degree (102.6°) and has a tendency to remain higher.

After the evening of the fifth day in patchy bronchopneumonia there is a rapid fall on the morning of the sixth day to 100°, then a gradual rise until the evening of the seventh day to 101°; in massive bronchopneumonia there is a daily fluctuating rise until the evening of the seventh day from 102.4° to 103.8°; in lobar pneumonia there is a daily fluctuating drop from 103° to 101°.

The pulse-rate in patchy bronchopneumonia from the first to the fifth day shows a steady and distinct rise from 100 to 128; in massive bronchopneumonia during the same period there is a fluctuating rise from 98 to 118; in lobar pneumonia the pulse-rate averaged about 110 without any marked degree of fluctuation until the evening of the fourth day, from then until the evening of the fifth day it rose to 120. From the fifth to the seventh day in patchy bronchopneumonia the pulse ranged about 120; in massive bronchopneumonia there was a steady rise from 112 to 130; in lobar pneumonia a daily fluctuation between 110 and 120.

The respiratory rate from the first to the seventh day in patchy bronchopneumonia shows a gradual fluctuation between 30 and 38; in massive bronchopneumonia a slow, gradual rise, with little fluctuation, from 24 to 40; in lobar pneumonia a gradual rise from 30 to 40.

(*c*) Blood picture. In the series of 105 cases of pneumonia blood counts were taken in only 24. In 6 cases of massive bronchopneumonia, irrespective of the day of the disease, the white blood count ranged between 9000 and 12,000; in 5 cases between 5000 and 9000; in 3 cases between 12,000 and 15,000; in one case above 24,000; and in one case under 5000. In 5 cases of lobar pneumonia the white blood count was between 5000 and 9000; in one case between 9000 and 12,000; and in one case above 24,000. No blood count was made in patchy bronchopneumonia.

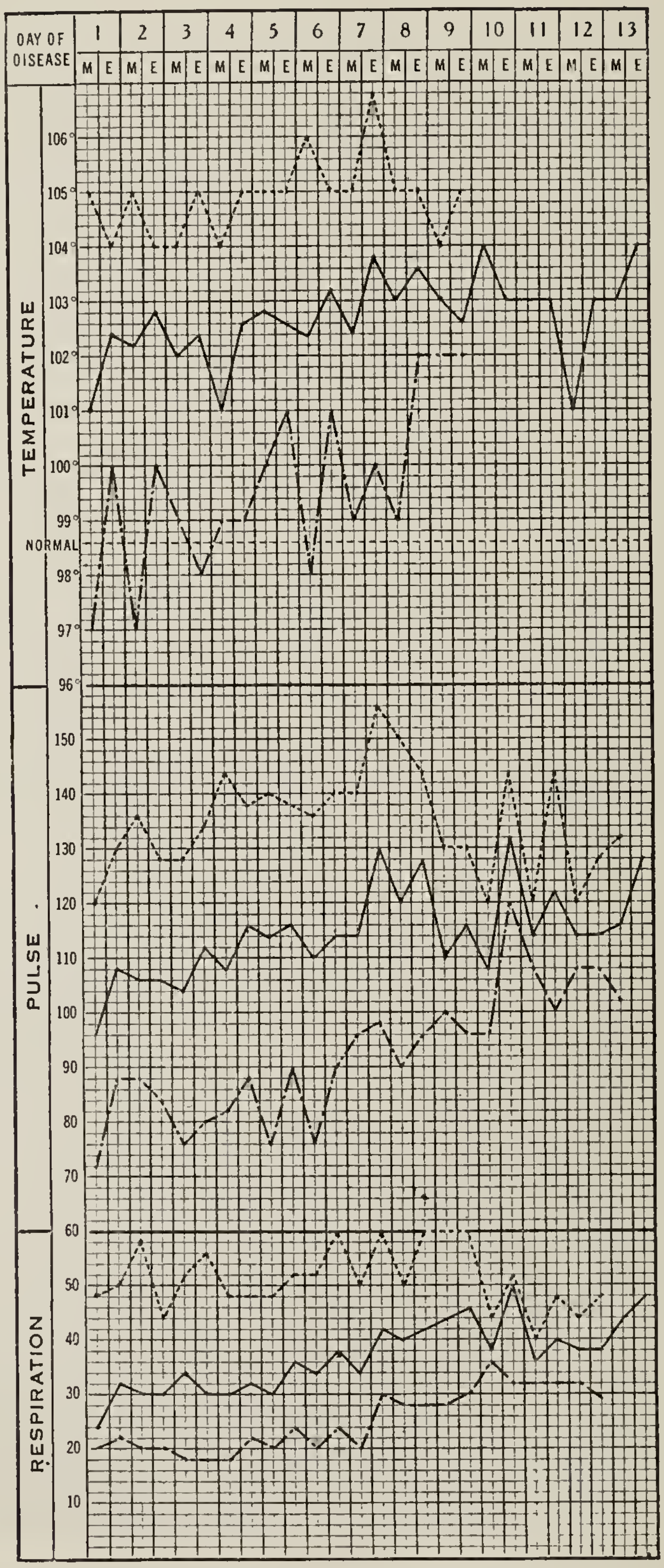

FIG. 111.—Massive bronchopneumonia (Composite chart).

No conclusions can be drawn from such a meager report.

A graphic chart of temperature, pulse and respiration is attached, showing the highest, the lowest and the mean rates in the different pathologic types of pneumonias, and in pneumonia caused by certain organisms.

IV. Etiology. In this series of 109 autopsies the heart blood was cultured in 94 cases. Of the 94 cases cultured the heart blood was positive 60 times.

It should be noted that the heart blood was positive in one case each for influenza bacillus and meningococcus, Type II, without the presence of other organisms.

It is to be noted, further, that every effort was made to obtain cultures from the smaller air passages, rather than from the bronchi. In the latter, upper respiratory organisms are likely to be aspirated during the terminal course of the disease, and therefore probably play no part in the existing diseased lungs.

V. Gross Classification of Pneumonias.

- Bronchopneumonia
  - Patchy
    - With serum.
    - With marked serum and blood.
  - Massive
    - With serum.
    - With marked serum and blood.

- Lobar pneumonia
  - Dry.
  - Bloody.

Gross Description. Patchy Bronchopneumonia with Small Amount of Serum: *Lung.* Not voluminous. The weight is slightly increased up to 400 gm. The pleura is pinkish gray, smooth and glistening when foci of consolidation are deeply placed in lung tissue; but when these areas are more superficial the pleura is studded with non-elevated, subserous, fiery red, irregularly shaped areas up to 3 mm. in diameter and frequently lusterless areas of thin, fibrinous exudate varying in size up to 1 cm. in diameter are seen especially overlying the superficially located nodules of consolidation. Frequently a few scattered, well-circumscribed, slightly depressed, reddish-brown areas are present under the pleura. These vary up to 2 cm. in diameter and are doughy and India rubber-like in consistency (atelectatic areas). Shot- or pea-sized areas of consolidation may or may not be felt in the substance of the lung, depending on whether these areas are superficially or deeply located. The organ pits slightly on pressure.

The section surface is moist, pinkish gray and mottled throughout with a few scattered elevated, smooth or finely granular, reddish-brown or reddish-gray areas varying in size up to 10 mm. Many such areas are located around the bronchioles. There are usually seen scattered throughout the section surface a few dark red, smooth, moist and glistening depressed areas up to 10 mm. in diameter (atelectatic patches). The section surface does not drip a markedly excessive amount of frothy material. The bronchioles in the affected areas contain variable amounts of yellowish-green purulent material, practically never blood-tinged.

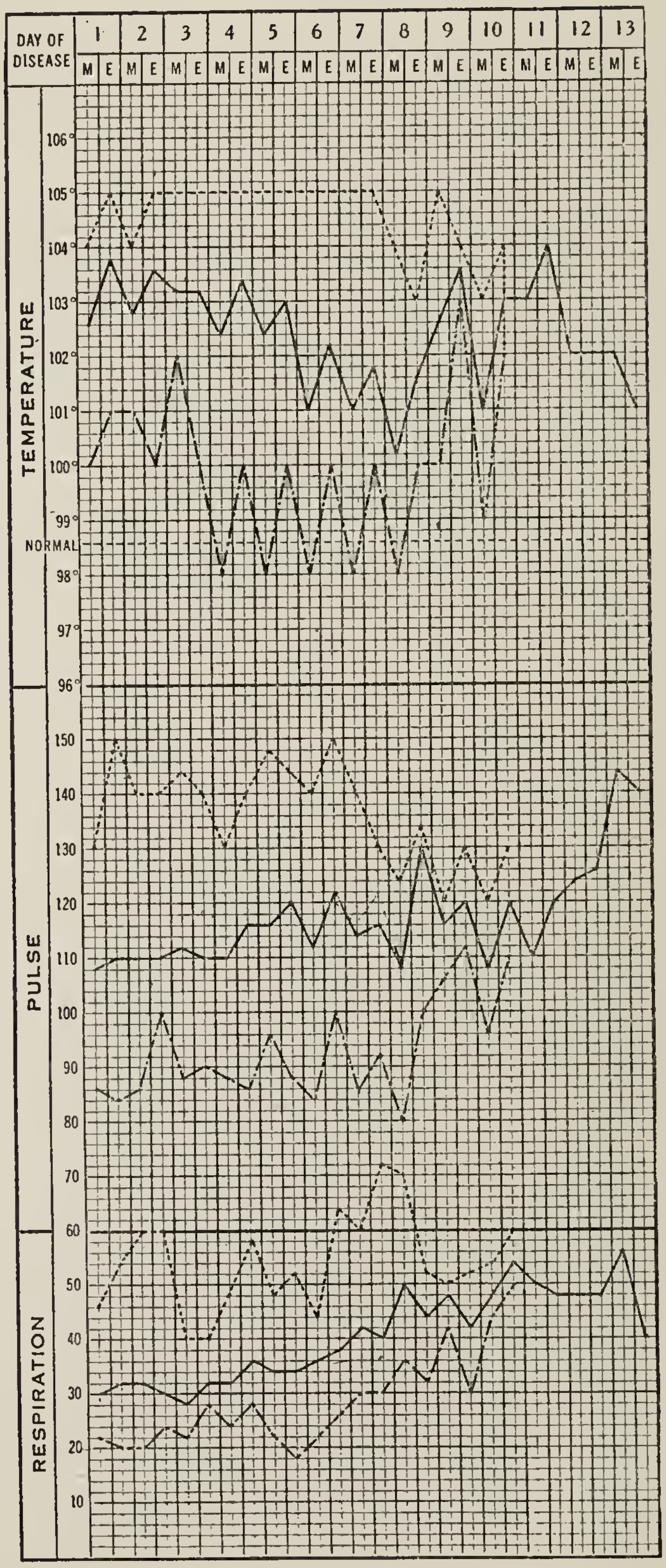

FIG. 112.—Lobar pneumonia. (Composite chart.)

PATCHY BRONCHOPNEUMONIA WITH AN EXCESSIVE AMOUNT OF BLOODY SERUM. The pleura presents the same picture as in the patchy type without marked serum. The organ is heavier, however, up to 500 gm., and is boggy—pitting on pressure, and, due to this edema, the consolidated areas are more difficult to palpate.

Section surface presents the same picture as in the type mentioned above, but in addition the whole organ, more especially the scattered areas of consolidation, drip an excessive amount of pinkish or reddish-brown (or in the pure hemolytic streptococcic cases often), fiery red, frothy material. When this fluid has escaped the elevated consolidated areas have a granular appearance, whereas before they were smooth. The bronchioles here often contain a blood-tinged purulent material.

MASSIVE BRONCHOPNEUMONIA WITH SERUM. *Lung.* Voluminous, frequently showing the imprint of the ribs. The weight is considerably increased averaging 800 to 1000 gms. The pleura frequently shows a fibrinous exudate, not always confined to that portion overlying the consolidation and varying in thickness from a frosted glass-like film to 3 mm.; it is usually firmly adherent. There are subpleural, fiery red areas up to 5 mm. in diameter. Uninvolved larger portions of the lung show slightly diminished crepitation and pit slightly on pressure. Involved areas are liver-like in consistency and vary in size greatly; some involving whole lobes and occasionally a whole lung, with the exception occasionally of a walnut-sized area at the apex or anteriorly. Here also are seen depressed, reddish-brown, doughy areas up to 2 cm. in diameter.

Section surface is moist, dark reddish gray in color, mottled with irregularly shaped and sized reddish-brown to reddish-blue, elevated, granular, non-crepitant, frequently confluent areas, with small areas of intervening lung tissue which is smooth and shows diminished crepitation, and is boggy. These areas have no definite relation to the bronchi other than that, regardless of how small an area may be, there is usually a bronchus of larger or smaller size near its center. Occasionally are seen depressed doughy areas of irregular size and shape which are smooth, moist, and beefy in appearance (atelectatic areas). The bronchi have a yellowish green, purulent material, often blood-tinged. The crepitating areas exude a slightly increased amount of frothy serous material, which also exudes from the consolidated areas but not in marked quantities.

MASSIVE BRONCHOPNEUMONIA WITH MARKED SERUM AND BLOOD: *Lung.* Voluminous as in the previous type and very heavy, reaching as high at times as 2000 gm. The pleura in this type shows the same changes as in the less moist type, but these changes are more constant. In addition, in this type, there occurs very frequently a serous effusion into the pleural cavity.

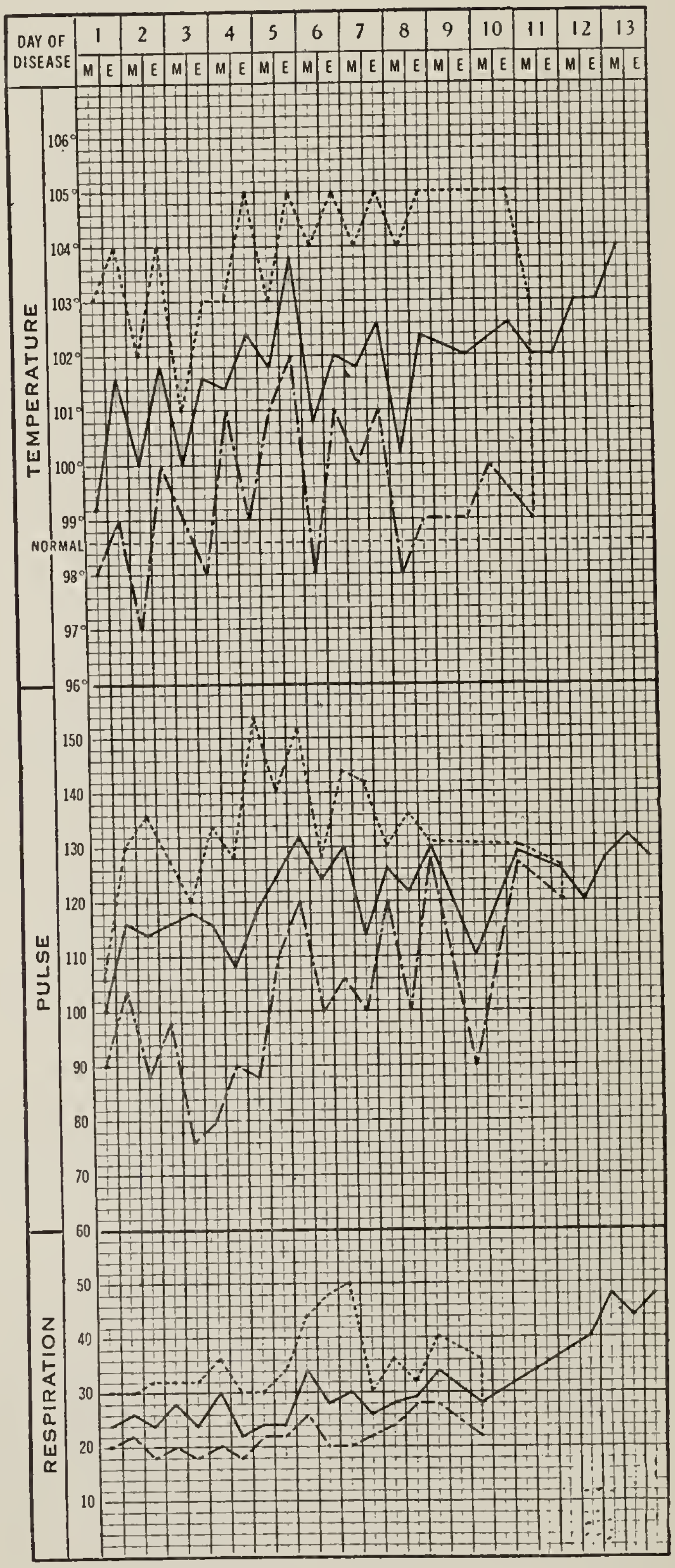

FIG. 113.—Hemolytic streptococcus. (Composite chart.)

On section the surface shows the same changes as the dry variety, but the whole section surface is dripping with great quantities of a bloody serous fluid with more or less admixed air, depending on the amount of remaining uninvolved lung tissue. The bronchial contents in this type are nearly uniformly bloody purulent material. When a section surface of this type is inverted the fluid pours out, leaving a pitted and coarsely granular surface.

Lobar Pneumonia (dry). This type is comparatively infrequent in this series, two cases only, and both occurred in pneumococcus infection superimposed on cases of gas inhalation. Only one shows fibrin in the pleura. The lungs grossly are those such as are described under lobar pneumonia, and the cut surface exudes little or no fluid material, except a semipultaceous yellowish material. No empyemas occurred in these and no serous fluid in the chest. The involvement here is mainly in the lower lobes, both cases showing one involved while one showed both. The bronchi are filled with purulent material.

Lobar Pneumonia (bloody). This type generally shows the same gross characteristics as those always described, except that the cut section exudes in most cases either a bloody serous fluid or a bloody semipultaceous material. The pleura shows fibrin in 24 out of 32 cases, and there were 7 cases of empyema out of the 32 cases. None show serous exudate in the pleura. The involvement is always more marked in the lower lobes, one or the other, or both, being entire in 29 cases. The bronchi are filled with yellowish-green semipultaceous purulent material.

VI. Microscopic Findings. (*a*) Patchy Bronchopneumonia with a small amount of serum. Total cases, 15.

Bronchi. The lumina of the bronchi were filled in most instances with leukocytes, mainly polymorphonuclears. In addition a few (3 cases) showed a small amount of serum, much fibrin, and many leukocytes, mainly polymorphonuclears. Still, 3 cases showed serum and red blood cells to be the predominant elements of inflammation.

The walls of the bronchi were thickened as the result of an inflammatory infiltration of fibrin and leukocytes, mainly polymorphonuclears (6 cases), and leukocytes, mainly polymorphonuclears, with the addition of marked capillary engorgement (4 cases). In the remaining 5 cases the walls showed in 2, marked capillary engorgement, with an inflammatory exudate of much serum and fibrin and many leukocytes, mainly polymorphonuclears; in 2, capillary engorgement with much fibrin and leukocytic infiltration, and in 1, marked capillary engorgement and serum infiltration.

Alveolar Sacs. The alveolar sacs adjacent to the bronchi and those extending for variable distances from same were filled in the majority of instances with an inflammatory exudate composed of serum, fibrin and leukocytes, mainly polymorphonuclears; these

Pneumonia.	Patchy broncho S	Patchy broncho B	Massive broncho S	Massive broncho B	Lobar D	Lobar B	Total	
Hemolytic streptococcus.	4	..	2	2	..	2	10	
Positive heart blood.	2	..	1	2	..	2	7	
Hemolytic streptococcus; pneumococcus, Type I.	..	..	..	1	..	..	1	
Positive heart blood.	..	..	..	1	..	..	1	
Hemolytic streptococcus; pneumococcus, Type IV.	..	..	..	1	..	..	1	
Positive heart blood.	..	..	..	1	..	..	1	
Hemolytic streptococcus; pneumococcus, type undetermined.	..	..	..	..	..	1	1	
Positive heart blood.	..	..	..	..	..	1	1	
Non-hemolytic streptococcus.	..	..	2	4	..	..	6	
Positive heart blood.	..	..	2	2	..	..	4	
Streptococcus, type undetermined.	2	3	..	1	..	..	6	
Positive heart blood.	..	..	..	1	..	..	1	Heart blood cultured in 5 cases only.
Non-hemolytic streptococcus; Bacillus influenza.	2	1	1	1	..	..	5	
Positive heart blood.	..	..	..	..	..	..	..	Heart blood cultured in 4 cases only.
Non-hemolytic streptococcus; pneumococcus.	..	..	3	1	..	4	8	
Positive heart blood.	..	..	2	1	..	4	7	
Pneumococcus, Type I.	..	..	..	2	..	1	3	
Positive heart blood.	..	..	..	1	..	1	2	
Pneumococcus, Type II.	..	..	..	..	..	4	4	
Positive heart blood.	..	..	..	..	..	4	4	
Pneumococcus, Type III.	1	..	..	1	..	6	8	
Positive heart blood.	1	..	..	1	..	3	5	
Pneumococcus, Type IV.	..	..	..	5	..	7	12	
Positive heart blood.	..	..	..	3	..	5	8	
Pneumococcus, type undetermined.	4	2	2	1	..	9	18	Heart blood cultured in
Positive heart blood.	3	1	2	1	..	5	12	Heart blood cultured in 9 cases only.
Pneumococcus after gas.	1	7	1	1	2	..	12	
Positive heart blood.	..	..	1	..	1	..	2	Heart blood cultured in 7 cases only.
Pneumococcus; Bacillus influenza.	1	..	2	3	..	..	6	
Positive heart blood.	..	..	1	2	..	..	3	
Bacillus influenza.	..	1	..	1	..	..	2	
Positive heart blood.	..	1	..	..	..	..	1	
Meningococcus; pneumococcus; Bacillus influenza.	..	..	..	1	..	..	1	
Meningococcus; streptococcus.	..	..	..	11	..	..	11	Heart blood (meningococcus only).
Influenza bacillus; Micrococcus flavus; Staphylococcus albus.	..	..	..	1	..	..	1	
Influenza bacillus; Staphylococcus aureus.	..	..	..	1	..	..	1	
Influenza bacillus; Streptococcus viridans; Staphylococcus aureus.	..	..	..	1	..	..	1	
Influenza bacillus; Staphylococcus albus; non-hemolytic streptococcus.	..	..	..	1	..	..	1	
Total, regardless of bacteriology.	15	14	13	31	2	34	109	

elements varied in preponderance in the different sacs, with the exception that the number of polymorphonuclears remained constantly high. In a number of instances (7 cases) the predominant elements of inflammation were serum and endothelial leukocytes, intermingled with a small amount of fibrin and a few red blood cells. In two cases the sacs were filled with leukocytes, mainly polymorphonuclears, associated with many small local hemorrhagic areas. Still in 2 cases was present a uniform distribution in the sacs of the elements—serum, fibrin, red blood cells, polymorphonuclears and endothelials, with small, scattered, local hemorrhages.

Throughout the entire series there were present focal hemorrhages 5 times and small abscesses 2 times.

Alveolar Walls. The majority of the alveolar walls were thickened as the result of capillary engorgement and infiltration of much fibrin and many leukocytes, mainly polymorphonuclears. In 3 cases the walls showed fibrin deposit and capillary engorgement alone.

Bloodvessels.

A. Local or segmental involvement of walls:

1. Subintimal infiltration of small amount of fibrin and few polymorphonuclears, cases 4.

2. Entire thickness: infiltrated with many polymorphonuclears and small amount of fibrin, case 1; infiltrated with red blood cells, case 1.

B. General, or involvement of entire circumference of walls:

1. Subintimal infiltration of polymorphonuclears, case 1.

2. Subintimal infiltration of red blood cells, case 1.

3. Entire wall infiltrated with many polymorphonuclears and small amount of fibrin, cases 2.

4. Perivascular infiltration of leukocytes, mainly polymorphonuclears, cases 11.

Perivascular Lymphatics. The lymphatic vessels were dilated in 9 cases; filled with leukocytes, mainly polymorphonuclears, in 5 cases; filled with red blood cells in 2 cases.

(*b*) Patchy Bronchopneumonia with serum and blood. Total cases, 14.

Bronchi. The lumen of the bronchi were filled in the majority of instances with an inflammatory exudate composed of a small amount of serum, a variable amount of fibrin and many leukocytes, mainly polymorphonuclears. In 4 cases, 3 showed the exudate to be of much serum and many red blood cells. Still in 2 cases the exudate was of endothelial leukocytes, mainly in 1, and polymorphonuclears and endothelials in the other.

The walls of the bronchi were thickened as the result of much fibrin and leukocytic infiltration, of polymorphonuclears mainly, in

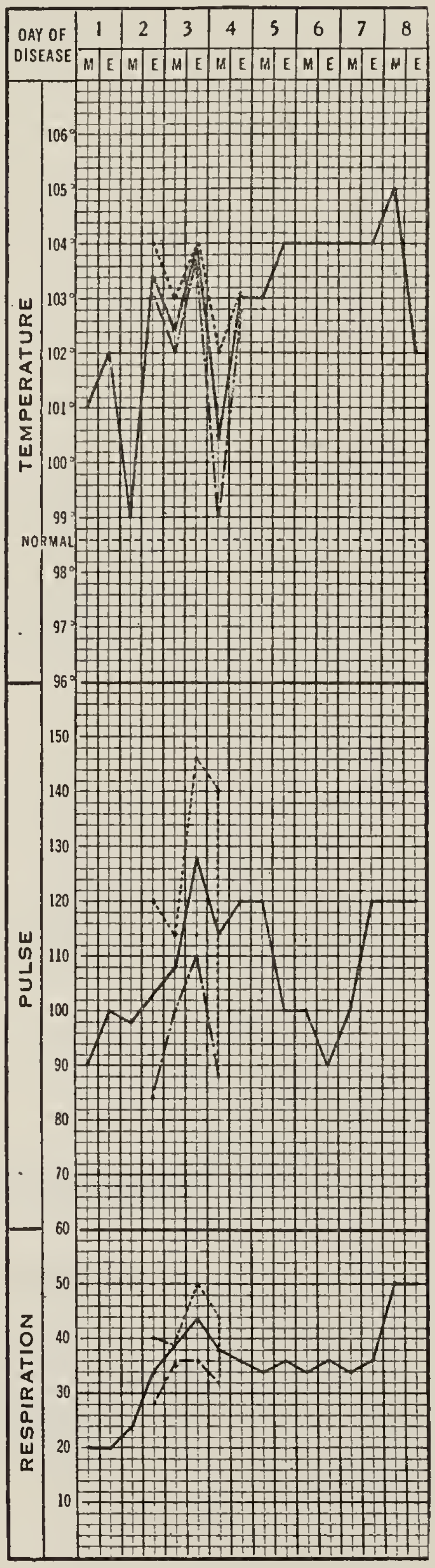

FIG. 114.—Non-hemolytic streptococcus. (Composite chart.)

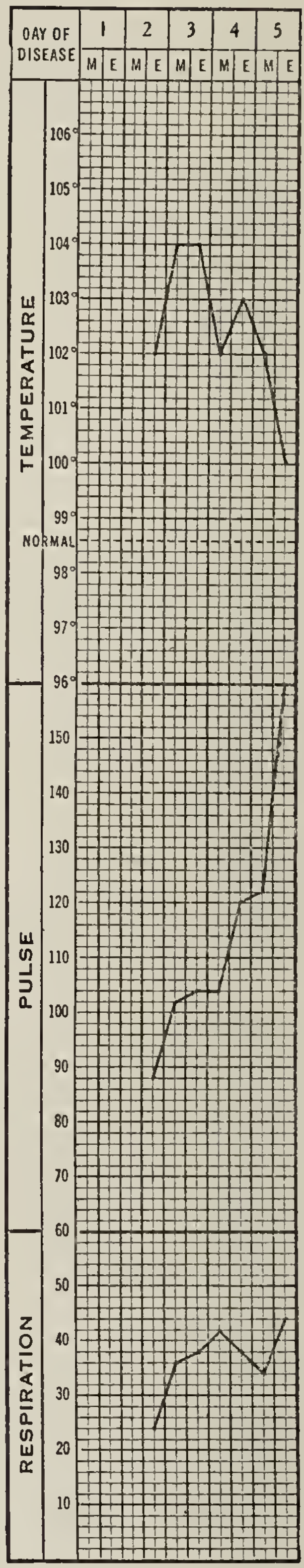

FIG. 115.—Streptococcus and meningococcus. (Composite chart.)

8 cases, 3 of which showed in addition capillary engorgement, and 1, serum infiltration. Still in 1 case the infiltration was composed of endothelial leukocytes and plasma cells.

Alveolar Sacs. The alveolar sacs adjacent to the bronchi and those extending for variable distance from same were filled in many instances with serum, fibrin and leukocytes, mainly polymorphonuclears; these elements varied in preponderance in the different sacs, especially so in the terminal alveoli, where the polymorphonuclears were constantly high. In half the cases the predominant elements of inflammation were serum and endothelial leukocytes intermingled with a small amount of fibrin and a few red blood cells. In one such case there were present small hemorrhagic areas.

Throughout the entire series there were present 1 case with focal hemorrhages, 1 case with small abscesses and 1 case with small infarcts. In 1 case were found a few healing miliary tubercles.

Alveolar Walls. The alveolar walls were thickened as the result of in 1 case much fibrin deposit and capillary engorgement; in 2 cases, fibrin and leukocytes, mainly polymorphonuclears; in 2 cases, endothelial leukocytic infiltration and capillary engorgement; and in 1 case much fibrin deposit.

Bloodvessels. A. Local or segmental involvement of walls:

1. Subintimal infiltration of small amount of fibrin, case 1.
2. Subintimal edema, case 1.
3. Entire thickness infiltrated with polymorphonuclears and small amount of fibrin, case 1.

B. General or involvement of entire circumference of walls:

1. Subintimal infiltration of much fibrin, case 1.
2. Entire wall infiltrated with many polymorphonuclears and small amount of fibrin, case 1.
3. Entire wall infiltrated with plasma cells and polymorphonuclears, case 1.
4. Perivascular infiltration of leukocytes, mainly polymorphonuclears, cases 4.

Perivascular Lymphatics. The lymphatic vessels were dilated in 9 cases; filled with polymorphonuclears in 3 cases; filled with endothelials in 1 case; and filled with red blood cells in 2 cases.

(*c*) Massive Bronchopneumonia with serum: Total cases, 13.

Bronchi. The lumina of the bronchi were filled in the majority of cases (8 cases) with leukocytes, mainly polymorphonuclears. In 3 cases, the lumen was filled with much serum and many red blood cells. In 1 case, a small amount of serum, much fibrin, and many leukocytes, mainly polymorphonuclears, were the predominant elements.

The walls of the bronchi, in most instances, were infiltrated with some serum, much fibrin, and many leukocytes, mainly polymorphonuclears. In a few cases, the capillaries were engorged.

Alveolar Sacs. The alveolar cells in half of the cases were filled with an inflammatory exudate composed of serum, fibrin, and leukocytes, mainly polymorphonuclears; the elements varying in preponderance in the different sacs. In the remaining half of the cases, the elements of inflammation varied markedly in their various combinations.

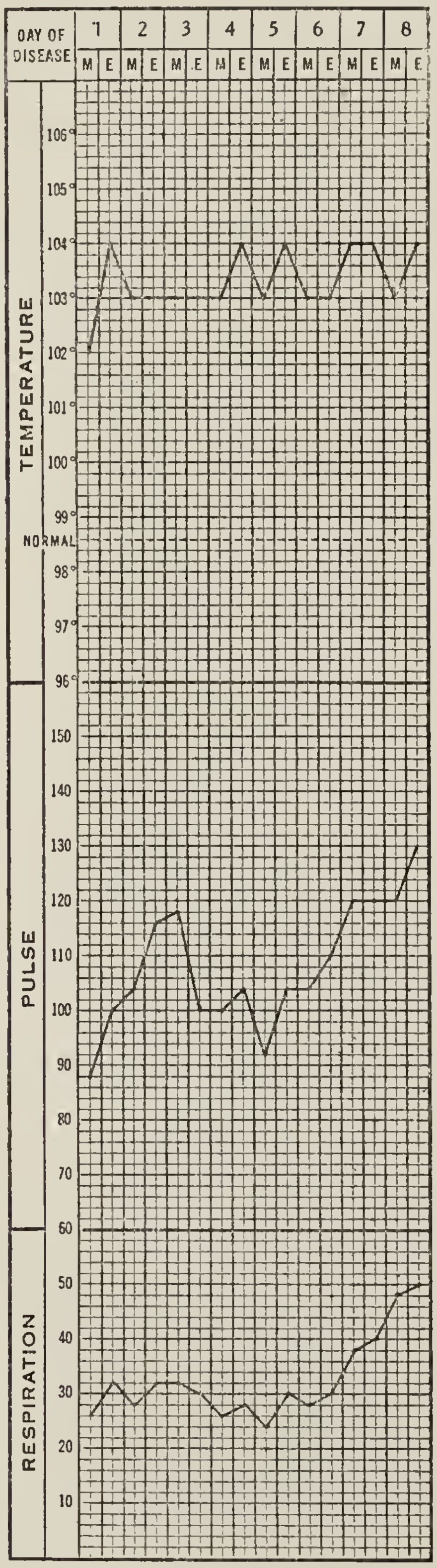

FIG. 116.—Influenza and streptococcus.

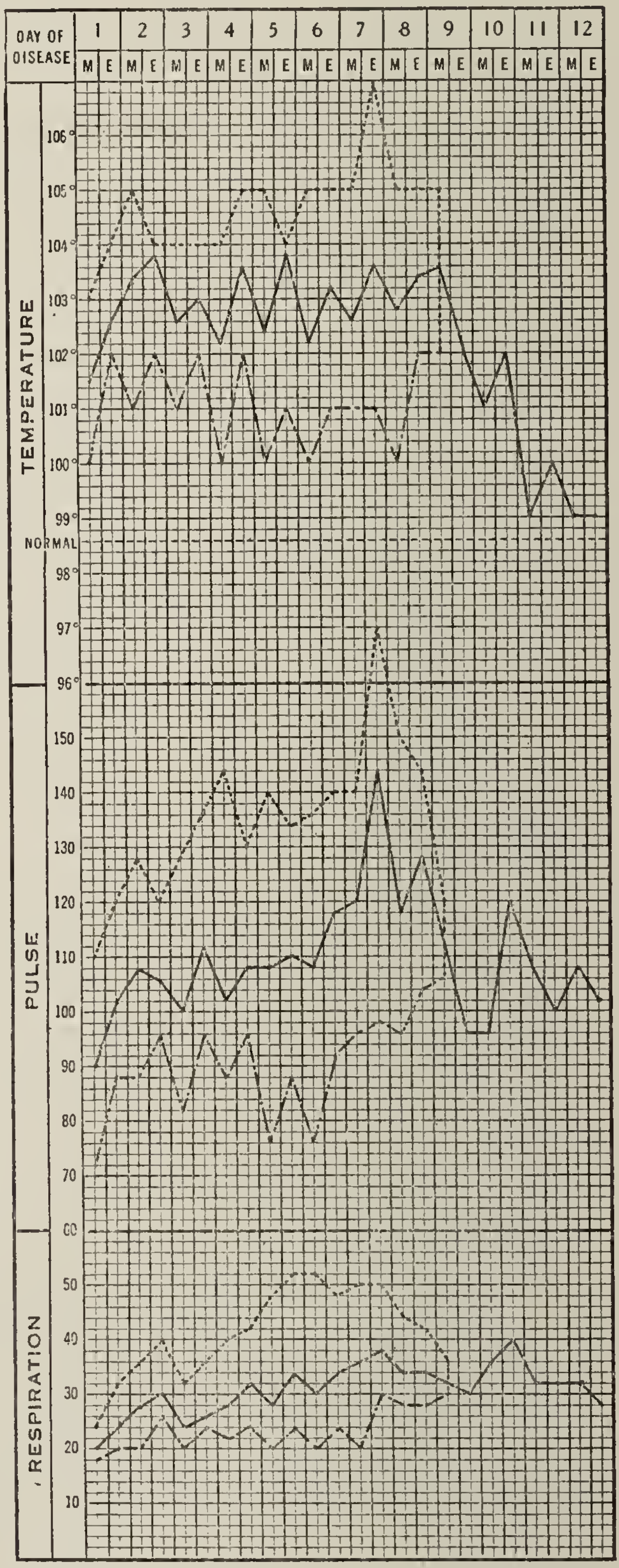

FIG. 117.—Non-hemolytic streptococcus and pneumococcus.

In two cases, the sacs were filled with much serum and fibrin, many red blood cells and endothelial leukocytes. The terminal sacs were filled with polymorphonuclears mainly; in addition, small focal hemorrhages were present. In 2 cases, serum and endothelial leukocytes admixed with a few red blood cells and a small amount of fibrin were the prevailing elements of inflammation. In 1 case, much fibrin, few red blood cells, and a small amount of serum, and a few small local hemorrhages were the microscopical findings. Still in 1 case, the predominant element was polymorphonuclears. Here, too, small focal hemorrhages were present.

In all the cases of this series, the sacs around the bronchi were mainly involved; however, there were scattered collections of alveolar sacs involved, which bore no relation to the bronchi, seen in the section. Nevertheless, they can be looked upon in many instances as terminal sac involvement.

In the series, 3 cases showed scattered small focal hemorrhages, and 1 case showed small abscesses.

Alveolar Walls. The alveolar walls showed capillary engorgement in 5 cases. In 2 cases, the walls were thickened as the result of capillary engorgement and much fibrin infiltration. In three cases, the walls were thickened as the result of much fibrin and leukocytic infiltration of polymorphonuclears.

Bloodvessels. A. Local or segmental involvement of walls:

1. Subintimal infiltration of an occasional polymorphonuclear and small amount of fibrin, cases 2.
2. Entire thickness infiltrated with polymorphonuclears, case 1.

B. General or involvement of entire circumference of walls:

1. Subintimal infiltration of polymorphonuclears, case 1.
2. Entire wall infiltrated with polymorphonuclears, case 1.
3. Perivascular infiltration with polymorphonuclears, cases 5.

Perivascular Lymphatics. The lymphatic vessels were dilated in 7 cases; filled with polymorphonuclears in 1 case; filled with endothelials in 1 case; and filled with red blood cells in 2 cases.

(*d*) Massive Bronchopneumonia with serum and blood: Total cases 29.

Bronchi. The lumina of the bronchi were filled in the majority of cases (21 cases) with leukocytes, mainly polymorphonuclears, and a small amount of fibrin. In 14 cases the predominant elements of inflammation were serum and red blood cells.

The wall of the bronchi was infiltrated in 17 cases with many leukocytes, mainly polymorphonuclears, and much fibrin. In 6 cases, capillary engorgement was combined with leukocytic infiltration. In 7 cases, serum and capillary engorgement were the principal involvement of the walls. Still in 2 cases, the walls were infiltrated with serum, fibrin, and leukocytes, mainly polymorphonuclears, combined with marked capillary engorgement.

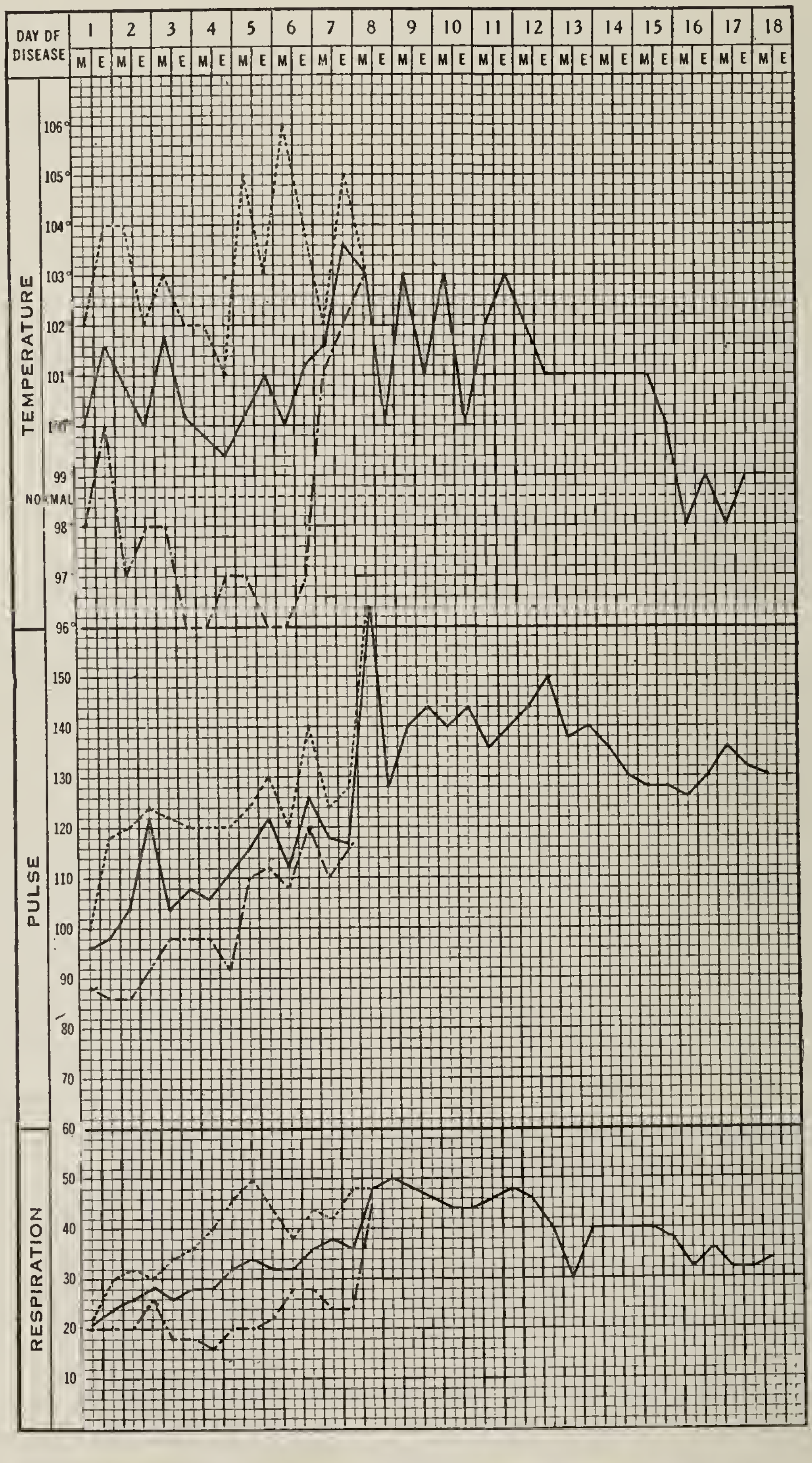

FIG. 118.—Streptococcus; type undetermined.

Alveolar Sacs. The alveolar cells in the majority of cases (23 cases) were filled with an inflammatory exudate composed of serum, fibrin, and leukocytes, mainly polymorphonuclears; the elements varied in preponderance in the different collections of sacs. The next most frequent combination of elements (11 cases) was much serum, many leukocytes, mainly polymorphonuclears, endothelials, a small amount of fibrin, and a few red blood cells. Added to this combination are 8 cases in which the red blood cells were many instead of being few.

In the entire series, small local hemorrhages were present in 17 cases; small infarcts in 2 cases; and small abscesses in 1 case.

Alveolar Walls. The alveolar walls were thickened in 24 cases as the result of infiltration of fibrin, or a combination of fibrin, serum and leukocytes, mainly polymorphonuclears. In 2 cases, endothelials were the prevailing elements. Capillary engorgement was prominent in 26 cases.

Bloodvessels. A. Local or segmental involvement of walls:

1. Subintimal infiltration of small amount of fibrin and few polymorphonuclears, cases 4.
2. Subintimal infiltration of fibrin, cases 3.
3. Subintimal infiltration of red blood cells, case 1.
4. Entire wall infiltrated with small amount of fibrin and few polymorphonuclears, cases 4.

B. General or involvement of entire circumference of walls:

1. Subintimal infiltration of polymorphonuclears, cases 5.
2. Subintimal infiltration of fibrin, cases 4.
3. Entire wall infiltrated with fibrin and polymorphonuclears, cases, 5.
4. Perivascular infiltration of leukocytes, mainly polymorphonuclears, cases 12.
5. Thrombus, Case 1.

Perivascular Lymphatics. The lymphatic vessels were dilated in 15 cases; filled with polymorphonuclears in 9 cases; filled with endothelials in 2 cases; filled with red blood cells in 8 cases.

(*e*) Lobar Pneumonia—Bloody. Total cases 32.

Bronchi. The lumen of the bronchi was filled in the great majority of cases (27 cases) with leukocytes, mainly polymorphonuclears. However, in 8 cases, serum and red blood cells were added.

In all the cases, the walls were infiltrated with a combination of serum, fibrin, and leukocytes, mainly polymorphonuclears. The leukocytes were the predominant element in most cases. There was in addition to the inflammatory condition, capillary engorgement in 11 cases.

Alveolar Sacs. The greater number of the alveolar cells in 30 cases were filled with an inflammatory exudate composed of serum, fibrin, and many leukocytes, mainly polymorphonuclears. However, red blood cells and endothelial leukocytes entered into the inflammatory picture in 17 cases.

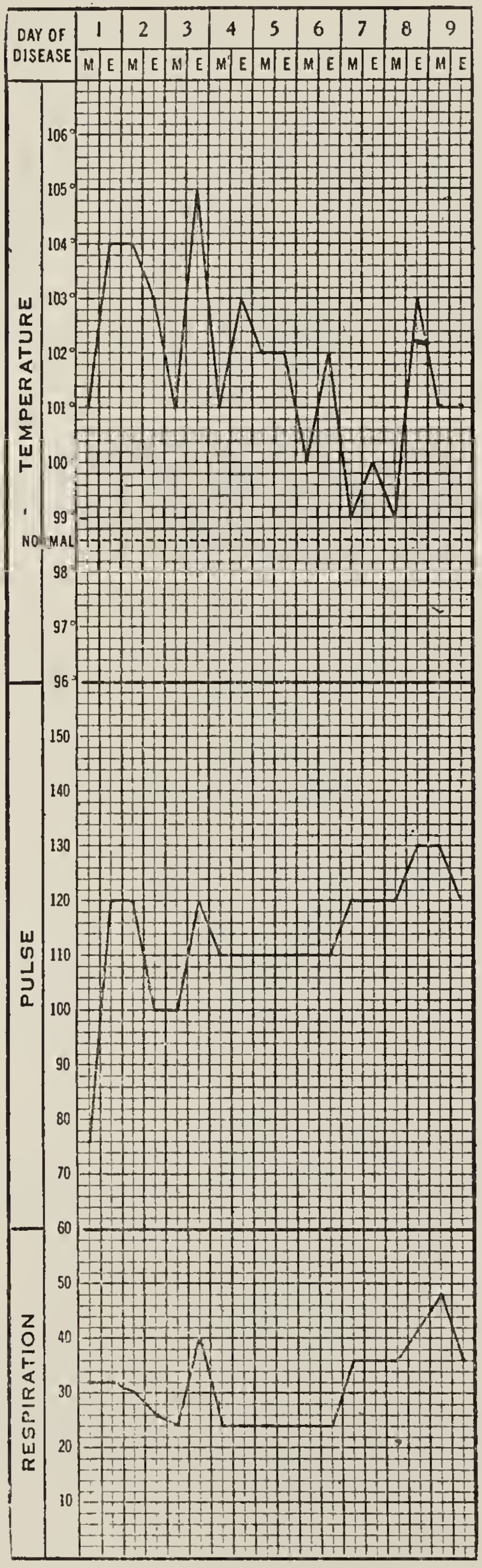

FIG. 119.—Pneumococcus; type I.

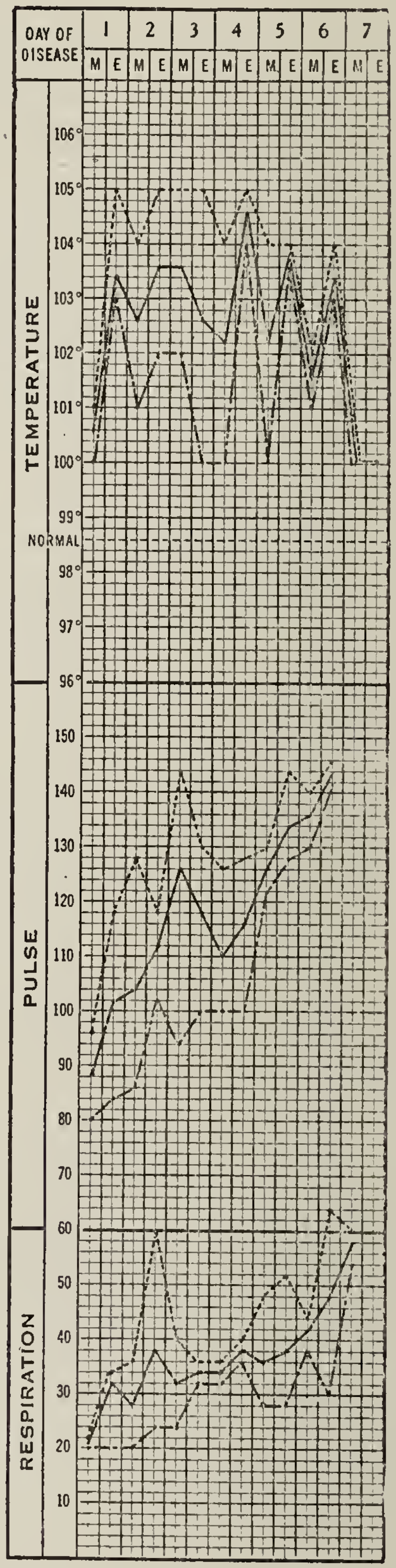

FIG. 120.—Pneumococcus; type II.

In the entire series small local hemorrhages were present in 17 cases; small infarcts in 2 cases; small abscesses in 1 case.

Alveolar Walls. The alveolar walls showed a variable amount of fibrin and leukocytic infiltration in 23 cases, and capillary engorgement in 21 cases. In 1 case, the predominant element of infiltration was endothelials.

Bloodvessels. A. Local or segmental involvement of walls:

1. Subintimal infiltration of small amount of fibrin and polymorphonuclears, cases 17.
2. Subintimal infiltration of small amount of fibrin, cases 3.
3. Entire wall infiltrated with polymorphonuclears mainly, cases 3.

B. General or involvement of entire circumference of wall:

1. Subintimal infiltration of polymorphonuclears, cases 12.
2. Subintimal infiltration of fibrin, cases 2.
3. Entire wall infiltrated with polymorphonuclears, cases 5.
4. Perivascular infiltration of polymorphonuclears, cases 15.
5. Thrombus, cases 2.

Perivascular Lymphatics. The lymphatic vessels were dilated in 14 cases; filled with polymorphonuclears in 13 cases; filled with red blood cells in 6 cases; filled with endothelials in 1 case.

SUMMARY OF MICROSCOPIC FINDINGS. *Bronchi.* The lumina of the bronchi in patchy bronchopneumonia without much serum, massive bronchopneumonia serum, and lobar pneumonia were filled —between 70 and 85 per cent. of cases with polymorphonuclears mainly; whereas the lumina of the bronchi in patchy bronchopneumonia with serum and blood and massive bronchopneumonia with serum and blood were filled, between 50 and 70 per cent. of cases with polymorphonuclears mainly. In the remaining cases the bronchi were filled with much serum, many red blood cells, and few leukocytes.

The walls of the bronchi showed capillary engorgement and were infiltrated with a small amount of serum and fibrin, few red blood cells, and many leukocytes, mainly polymorphonuclears in patchy bronchopneumonia without serum—90 per cent of cases (40 per cent. of cases without capillary engorgement); in patchy bronchopneumonia with serum and blood—83 per cent. of cases (40 per cent. of cases without capillary engorgement); in massive bronchopneumonia with blood and serum 100 per cent. of cases (50 per cent. of cases without capillary engorgement); in lobar pneumonia 100 per cent. of cases (70 per cent. of cases without capillary engorgement).

Alveolar Sacs. Scattered areas in which there was a variable distribution of serum, fibrin, red blood cells, endothelials, and a few polymorphonuclears. Patchy bronchopneumonia without serum, 80 per cent. of cases; patchy bronchopneumonia with serum and blood, 70 per cent. of cases; massive bronchopneumonia with serum, 54 per cent. of cases; massive bronchopneumonia with serum and blood, 89 per cent. of cases; lobar pneumonia, 60 per cent.

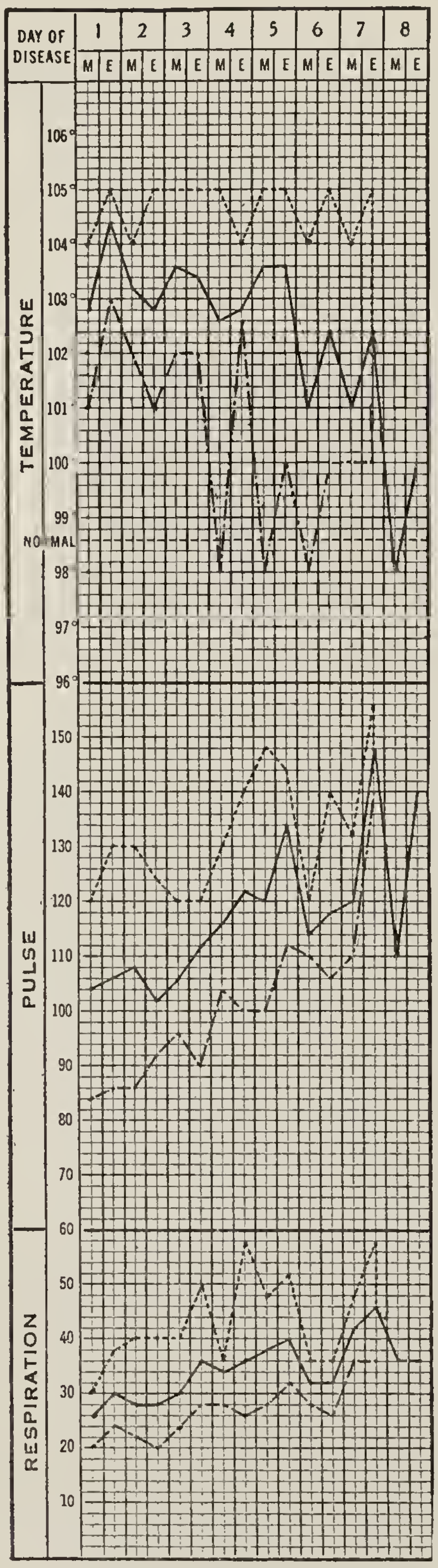

Fig. 121.—Pneumococcus; type III.

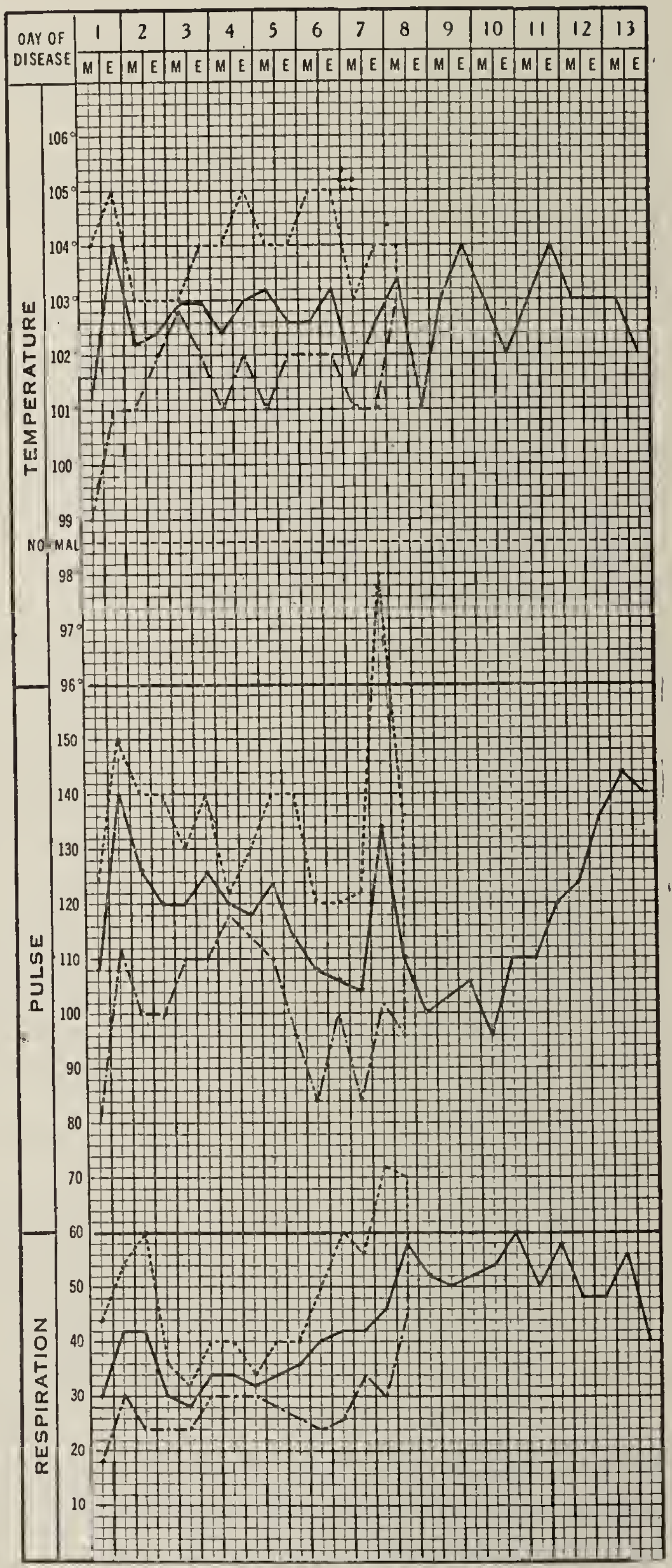

Fig. 122.—Pneumococcus; type IV.

Gross pathology, 105 cases.	Pathological types regardless of bacteriology.										Pneumonia.		
	Bronchopneumonia.								Lobar pneumonia.		Broncho.		Lobar.
	Patchy.				Massive.						Patchy.	Massive.	
	Serum.	Serum; blood.	Abscess.	Infarct.	Serum.	Serum; blood.	Abscess.	Infarct.	Dry.	Bloody.	29 cases	42 cases	34 cases
Left lung (entire)	5	2	..	..	4	12	..	..	..	5	7	16	5
Upper lobe (entire)	2	1	..	..	1	3	..	..	1	5	3	4	2
Upper third	1	1	..	..	1	3	..	..	..	5	2	4	6
Middle third	..	..	..	..	1	2	..	..	..	6	1	3	6
Lower third	..	..	..	..	..	3	..	..	..	5	..	3	5
Lower lobe (entire)	3	6	1	..	5	13	..	1	1	12	9	18	13
Upper third	1	1	..	..	1	1	..	..	2	4	2	2	6
Middle third	1	..	..	..	..	..	..	..	2	3	1	..	5
Lower third	..	..	..	..	..	1	..	..	..	..	..	1	1
Right lung (entire)	5	..	..	..	6	12	1	..	..	6	5	18	6
Upper lobe (entire)	2	5	..	..	2	12	..	..	..	3	7	14	3
Upper third	3	..	..	..	3	2	..	..	2	11	3	5	13
Middle third	..	..	..	..	1	2	..	..	1	9	..	3	10
Lower third	..	..	..	..	..	..	..	..	..	1	..	..	1
Middle lobe (entire)	3	3	..	..	..	10	..	1	..	6	6	10	6
Upper third	1	1	..	..	..	1	..	..	1	5	2	1	6
Middle third	1	1	..	..	..	1	..	..	..	3	7	1	3
Lower third	..	..	..	..	..	..	..	..	..	2	..	..	2
Lower lobe (entire)	4	10	..	..	4	14	..	1	2	17	14	18	19
Upper third	..	..	..	..	2	..	..		..	4	..	2	4
Middle third	..	..	..	..	1	1	..	..	..	4	..	2	4
Lower third	..	..	..	..	..	1	..	..	..	..	..	1	
Bilateral involvement:													
Pleura	..	..	..	..	..	8	..	..	..	4			
Patchy congestion													
Fibrin													
Serous fluid													
Purulent fluid													

Scattered areas in which many air sacs were filled with serum, fibrin, and many polymorphonuclears. Patchy bronchopneumonia without serum, 70 per cent. of cases; patchy bronchopneumonia with serum and blood, 64 per cent. of cases; massive bronchopneumonia with serum, 54 per cent. of cases; massive bronchopneumonia with serum and blood, 83 per cent. of cases; lobar pneumonia bloody, 94 per cent. of cases.

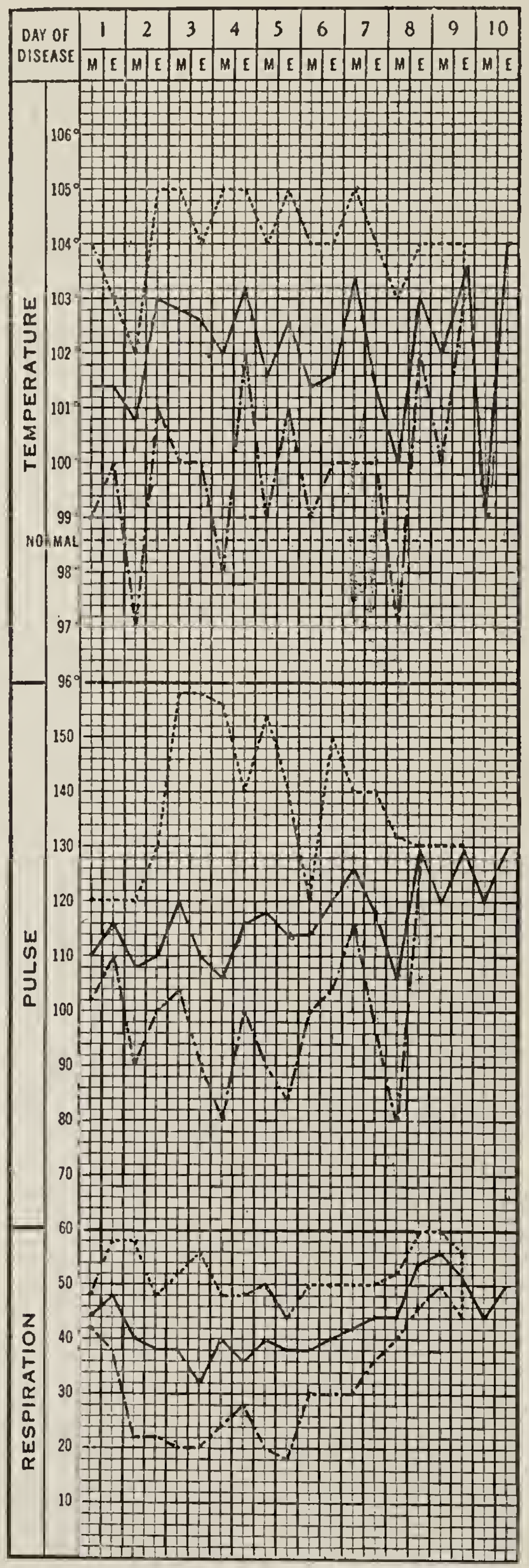

FIG. 123.—Pneumococcus; undetermined.

Alveolar Walls. The walls are thickened as the result of serum, fibrin, polymorphonuclears, and endothelial infiltration. Patchy bronchopneumonia without serum, 66 per cent. of cases; patchy bronchopneumonia with serum and blood, 40 per cent. of cases; massive bronchopneumonia with serum, 40 per cent. of cases; massive bronchopneumonia with serum and blood, 83 per cent. of cases.

VII. Blood and lymph vascular involvement by certain organisms, regardless of gross classification.

In classifying the gross lesions of pneumonias under the causative organisms, we found conspicuous microscopical changes in the bloodvessels that are characteristic in a way for certain groups of organisms.

Bloodvessels.	Patchy broncho.		Massive broncho.		Lobar, per cent.
	Without serum. per cent.	Blood and serum, per cent.	With serum. per cent.	Blood and serum, per cent.	
Local subintimal infiltration . . .	40	18	7	29	62
Local infiltration of entire wall . .	6	6	6	17	9
General subintimal infiltration . .	14	6	6	31	43
General infiltration of entire wall (panarteritis)	13	12	6	17	15
Perivascular infiltration of leukocytes	74	28	38	41	49
Perivascular lymphatics:					
Dilated	60	69	40	52	44
Lumen containing variable number of polymorphonuclears	33	21	7	31	40
Lumen containing variable number of endothelials	0	7	7	7	3
Lumen containing variable number of red blood cells	13	14	17	27	19

Namely: In cases in which hemolytic streptococcus alone was the causative organism, the perivascular infiltration of leukocytes —mainly polymorphonuclears—was present in all of the cases in which there was a patchy bronchopneumonia, but absent in the majority of cases in which there was a massive bronchopneumonia. In half of these cases of massive bronchopneumonia, there was present a general subintimal infiltration of fibrin and polymorphonuclears.

In a great majority of cases of all types of pneumonia in which non-hemolytic streptococcus alone was the causative organism, there was present a perivascular infiltration of leukocytes—mainly polymorphonuclears. In only one instance was there a subintimal inflammation; even then, the involvement was localized in one-half of the circumference of the intima. However, non-hemolytic streptococcus in combination with other organisms, especially with

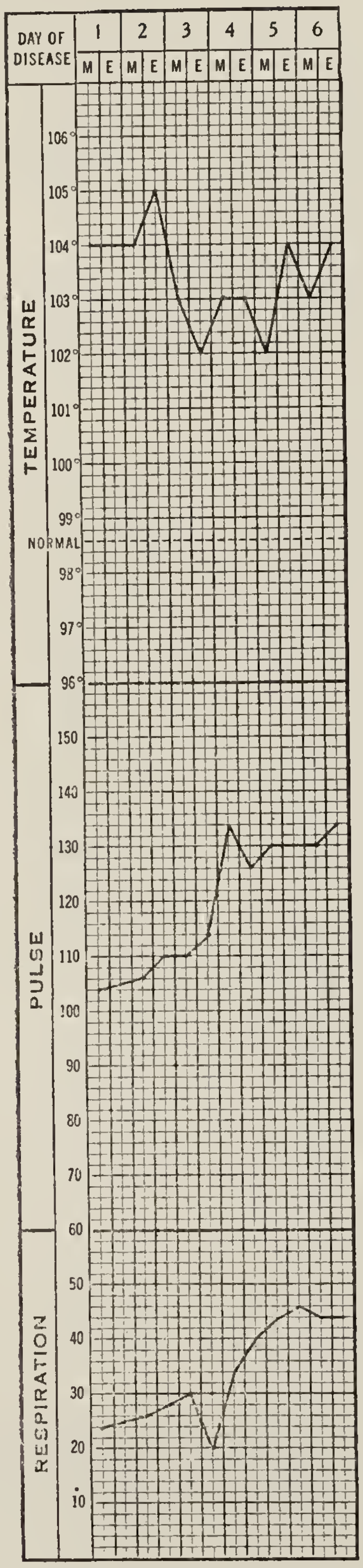

FIG. 124.—Pneumococcus and influenza.

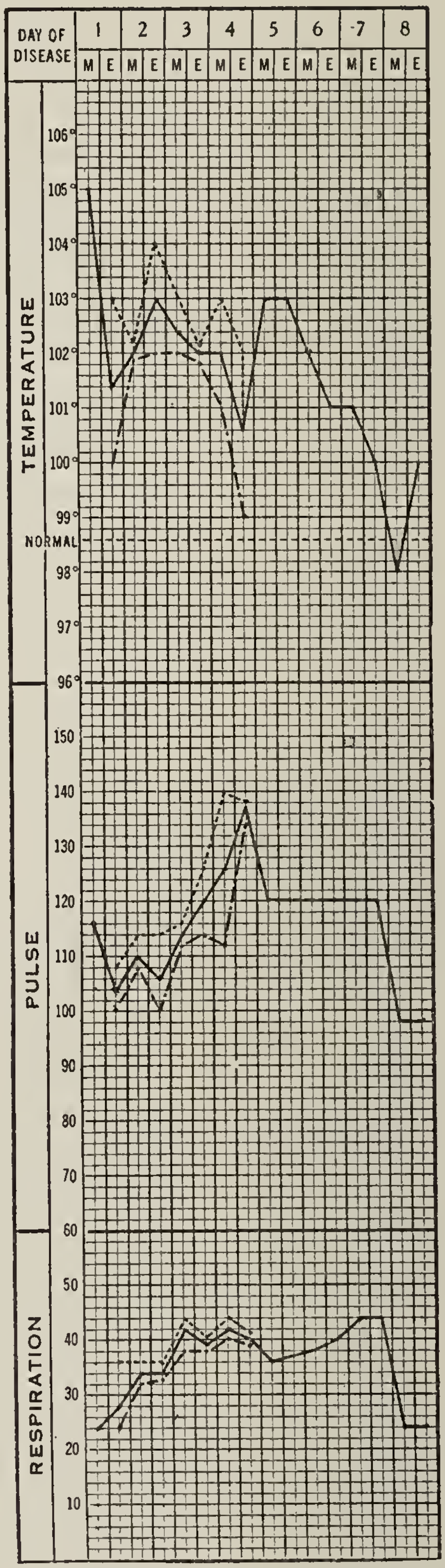

FIG. 125.—Influenza bacillus.

pneumococcus, shows the reverse to be true; that is to say, perivascular infiltration was infrequent, while subintimal infiltration of fibrin and polymorphonuclears occurred in the majority of cases. This is due in all probability, as will be shown later, to the associated organisms, namely pneumococcus.

In all cases of streptococcus, type undetermined, not associated with other organisms, there was present a perivascular infiltration in 100 per cent. of cases; in no instance was there present a subintimal involvement.

In the cases of pneumonias in which the pneumococcus, regardless of type, was the causative organism, there was present in the majority of instances either a subintimal infiltration of fibrin and polymorphonuclears, or an infiltration of the entire wall with serum, fibrin, and polymorphonuclears. With the exception of pneumococcus type III, only a small percentage of cases showed a perivascular infiltration. However, in pneumococcus type III, there was present in 100 per cent. of the cases a perivascular infiltration of polymorphonuclears mainly.

In the 2 cases in which the influenza bacillus alone was present, the bloodvessels showed a subintimal infiltration of fibrin and polymorphonuclears. There was no evidence of a perivascular infiltration.

Influenza bacillus in combination with non-hemolytic streptococcus showed an equal percentage of involvement between the subintimal and perivascular spaces. On the other hand, combination of influenza bacillus and pneumococcus, type undetermined, showed in the majority of cases a subintimal infiltration of fibrin.

In the combination of meningococcus, influenza bacillus, and pneumococcus, there was a general subintimal infiltration of much fibrin. Whereas, in the combination of meningococcus and streptococcus, there was a marked perivascular infiltration with subintimal change.

The perivascular lymphatic engorgement with polymorphonuclears or red blood cells was constantly high in pneumonias due to pneumococcus type IV, pneumococcus type undetermined and influenza bacillus, and hemolytic streptococcus, regardless of the gross lesion present. Otherwise, the perivascular lymphatic involvement was less than 50 per cent., regardless of the organisms or the gross lesion present. The latter applies to pneumonias complicated with acute pleuritis.

Positive heart blood found at autopsy apparently has no bearing upon the mode or severity of blood vascular involvement.

The hyalin-like material that was so frequently found in the subintimal spaces of bloodvessels and in the walls of the infundibular portion of the air passages, in massive bronchopneumonia, has been correctly or incorrectly called fibrin. This material was found fairly constant in the areas already designated, regardless of the underlying causative organisms.

OBSERVATIONS ON EMPYEMA[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

At the meeting of the American Surgical Association, twenty-six years ago my father opened the discussion on empyema. He laid down the following six propositions for discussion:

1. "No operation is justifiable"
   (*a*) "unless the presence of pus is certain;"
   (*b*) "unless thorough treatment by medicinal agents, blisters, etc., has failed;"
   (*c*) "or unless the symptoms, dyspnea, etc., are so urgent as to demand immediate relief."
2. "The first operation should consist of simple aspiration with antiseptic precautions."
3. "That when the fluid has partially reaccumulated, as it almost certainly will do if purulent, incision and drainage should be practised."
4. "That drainage is best effected by making two openings, one at the lowest available point, and carrying a large drainage tube through the cavity from one opening to the other."
5. "That drainage should be supplemented by washing out the cavity with mild antiseptic fluids. When the lung has expanded and discharge has nearly ceased the tube should be shortened, the upper opening being allowed to heal and the

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[1] Read before the American Surgical Association, May, 1920. Reprinted from *Annals of Surgery*, 1920, ii, 12.

tube being then gradually withdrawn through the lower opening."

6. "That when the lung is so bound down by adhesions that it cannot expand, resection of one or more ribs should be practised (Estlander's operation, so called), in order to allow collapse of the chest wall and to promote healing by bringing the costal and visceral layers of the pleura into contact. The more extensive operations of Schede and Tillmans, while probably justifiable in exceptional cases, are not to be recommended for general employment."

The mortality following operation for empyema at that time averaged from 20 to 30 per cent., and such it still remained in the hands of the average surgeon during the generation which has elapsed since 1894. It is true that in most of the fatal cases the operation is not to blame for the fatality, but merely fails to save life; and that death is due to the underlying disease —empyema.

It is my object in this discussion to see, if possible, in what ways we have made an advance over the teachings and practices of a generation ago.

In the first place, I call attention to the *first proposition:* "That no operation is justifiable except under one of these conditions," namely, (*a*) that the presence of pus is certain, (*b*) that medical treatment has failed, or (*c*) that the symptoms are very urgent. Is anyone at present ready to go beyond this and adopt operation under any other circumstances? Do we not always receive our patients from the physician whose treatment has failed? True it is that the physician almost invariably has made one or more punctures of the chest, with or without success in locating pus, before he calls the surgeon into consultation; and that similarly even when pus has been found he is often loath to turn the patient over to the surgeon until he finds that the pus does reaccumulate; and even under the latter circumstances he may persist in the attempt to cure the patient without surgical assistance, by means of antiseptic injections into the pleural cavity. So eminent and able a surgeon as John B. Murphy, it will not be forgotten, advocated and practised injections of a 2 per cent. solution of formalin in glycerin for the cure of empyema; but no statistics of this treatment, showing its results, have ever been published, so far as I am aware. Nor has the treatment been generally accepted, though it must be acknowledged that in perhaps 2 or 3 per cent. of cases in which the first aspiration shows only sero-pus, not frank pus, it is possible that a cure may be secured either by repeated aspiration alone or by aspiration followed by injection of formalin-glycerin solution, flavin, etc. So much, therefore, for the contingency which renders operation proper when medical treatment has failed.

Now if the symptoms are very urgent, that is if there is extreme dyspnea, cyanosis, bulging interspaces, etc., everyone admits the propriety of aspirating the chest whether or not it is certain beforehand that pus, not serum, is the cause of the symptoms. Finally, if the presence of pus is certain, comparatively few surgeons deny the propriety of attempting to evacuate it.

But one may rejoin, "How is it possible to be certain that pus is present except by aspiration? Are the clinical history, the physical signs, the roentgen-ray findings and the symptoms always sufficient to render a diagnosis certain?" To this I think we must reply in the negative; but I submit we must at the same time recognize that there are cases in which the presence of pus is so nearly certain, even when it cannot be located by puncture, that it is not only justifiable but imperative for the surgeon to open the thorax, search for the encapsulated empyema and effect its evacuation and drainage.

Some years ago, before the Section on General Medicine of the College of Physicians of Philadelphia, I presented[1] a plea for exploratory thoracotomy in just such cases of encysted empyema as these—cases where repeated puncture failed to locate pus; where the physical signs were suggestive; and where the symptoms were urgent. Realizing that the patients would die of their empyema unless the pus were drained, it seemed better to me to run the risk of failure to relieve them by exploration, rather than to fold my surgical hands together in impotence, and reiterate to the physicians who called me as consultant, what I know they had heard before in similar circumstances from other surgeons, a request to the physician to find the pus, with the statement that then, and then *only* would the surgeon undertake to drain it! Some surgeons, I may add, are even more conservative than this, and even though pus has been found by puncture by the physician, refuse to undertake any operation unless they themselves succeed in finding pus by a puncture made on the operating table. To my mind, pus is pus, whether found by the physician or surgeon; and I believe if it is found within the pleura the chances that it will be absorbed and cause no further trouble are so very remote that unnecessary delay in resorting to operation is unjustifiable. In some cases, of course, it is possible that an undrained interlobar empyema will drain itself by rupture into a bronchus; but it has yet to be shown that this event improves the patient's chance of recovery. Of two cases in my series (Case 8 and 13) only one recovered.

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[1] "Surgical Experiences with Encapsulated Empyema and Abscess of the Lung; a Plea for Exploratory Thoracotomy," Medical and Surgical Reports of the Episcopal Hospital, Philadelphia, 1916, iv, 226.

In the report to which allusion has already been made, I recorded:

1. A case of empyema encapsulated between the upper and middle lobes of right lung, discharging through the bronchus; punctures negative; empyema found on exploratory operation; death from exhaustion and sepsis (Case 8 of the present series).
2. A case of empyema encysted between the upper and middle lobes of the right lung. Numerous punctures negative; empyema not located at exploratory operation, but later burst into drainage tract; ultimate recovery (Case 18 of the present series).
3. A case of empyema encysted between the left lung and the diaphragm. Punctures negative; empyema found on exploratory operation; recovery (Case 24).

4. A case of empyema encapsulated between the upper and middle lobes of the right lung. Puncture negative; empyema found on exploration; recovery (Case 25).

5. A case of empyema encapsulated between the left lung and pericardium. Punctures negative; empyema not found at exploratory operation; death eight days later from continuing sepsis (Case 26).

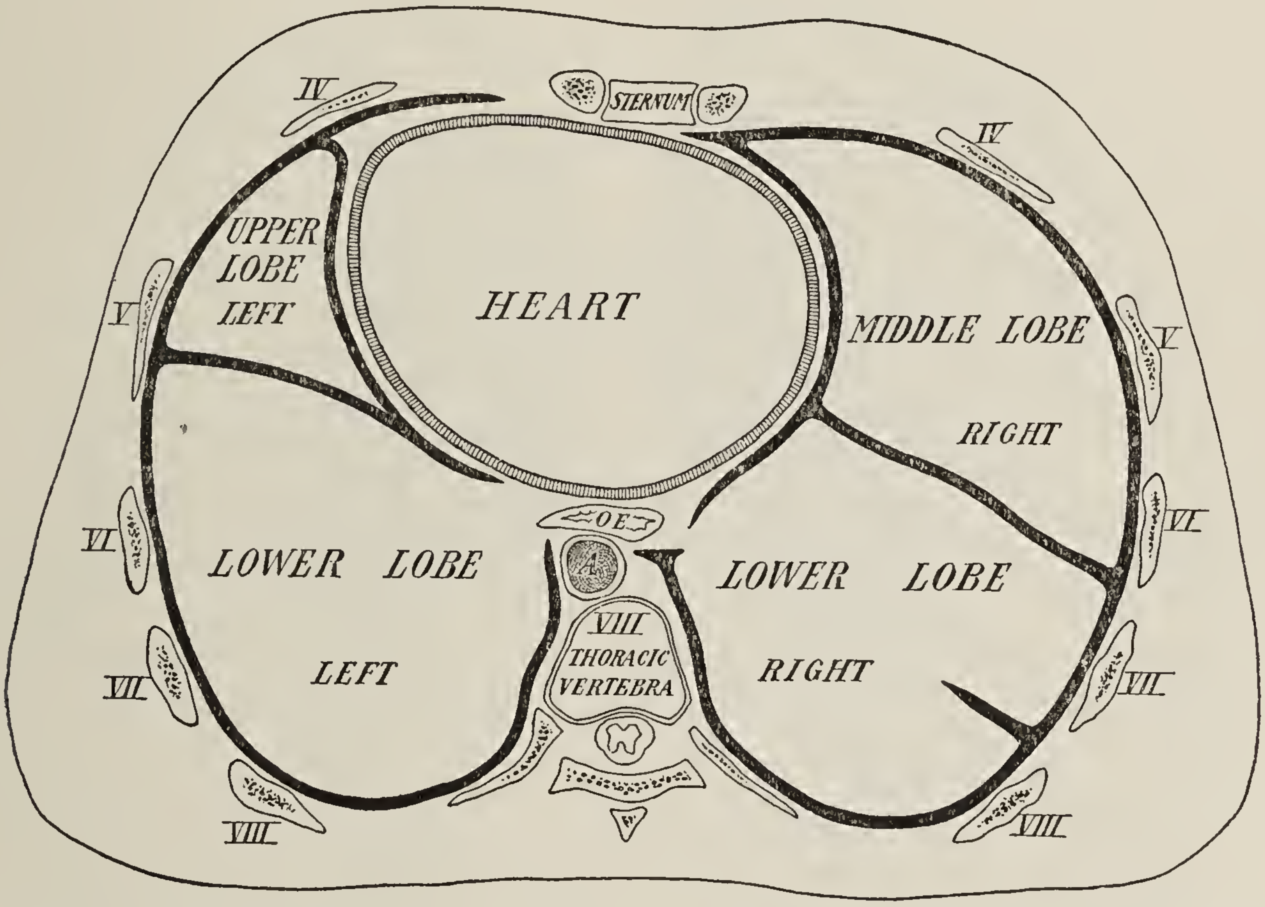
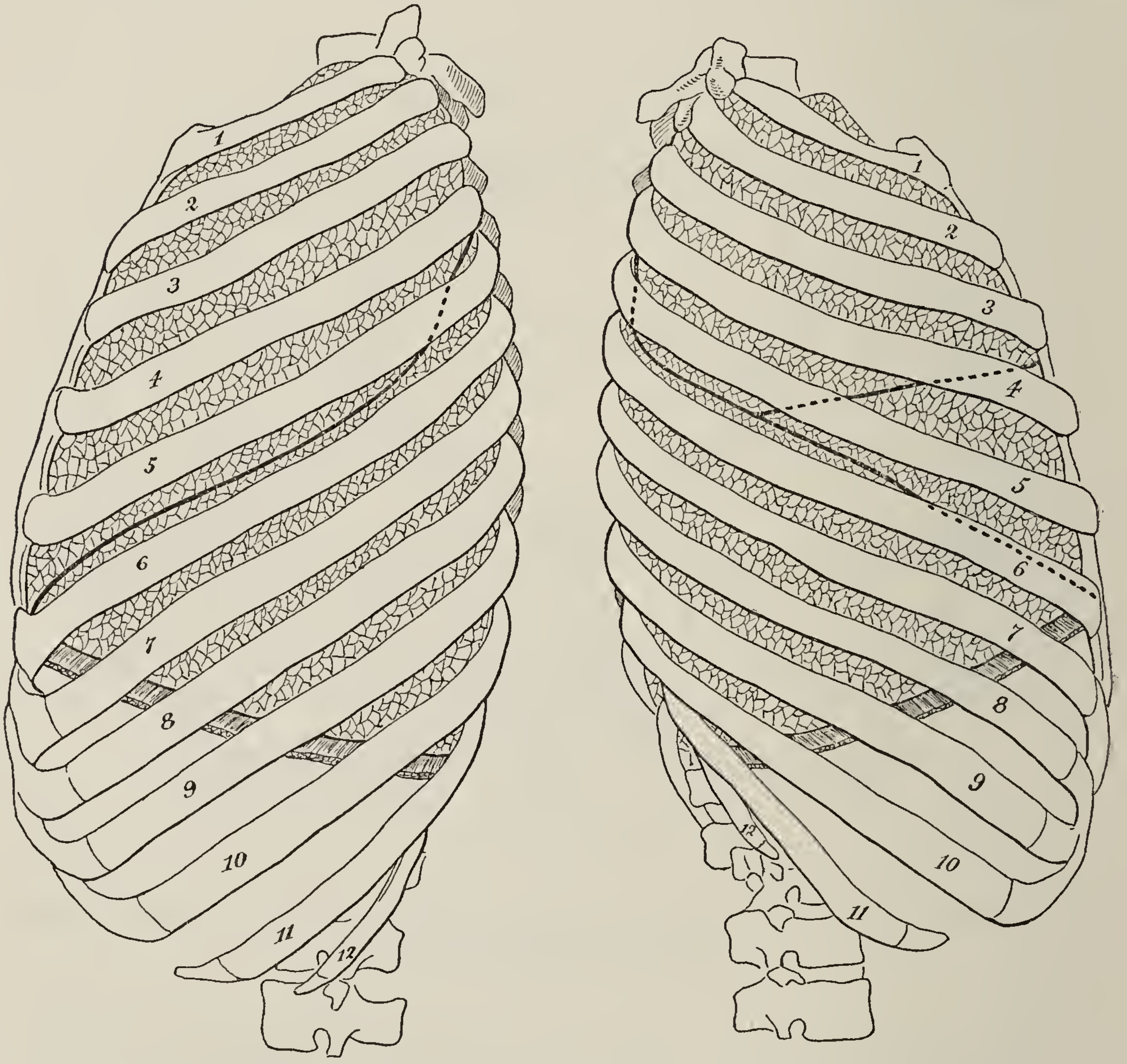


Fig. 126.—Cross-section of thorax at level of eighth thoracic vertebra. Pleural cavities outlined in black.

Now I think it is worthy of note that in the last-mentioned case had a roentgen-ray examination been available before operation, it is extremely probable that the empyema might have been located when the chest was opened; since roentgen-ray examination subsequent to operation (after necessary repairs to the roentgen-ray plant) showed distinctly the outlines of the abscess cavity (Fig. 142), and since autopsy showed that the interlobar dissection made at the time of the operation almost had reached the abscess (Fig. 141).

So that, with regard to the *first and second propositions* laid down twenty-six years ago, I think we must understand them as permitting and encouraging at the present time *exploratory thoracotomy,* when symptoms are urgent, medical measures have failed, repeated punctures are negative and yet the presence of pus seems certain. Under these circumstances I have opened the thorax for exploration on six occasions (Cases 8, 18, 24, 25, 26, 30), and except in the two cases just mentioned (Cases 18 and 26) succeeded in finding and draining the empyema every time; and while only 3 of these patients recovered, I am sure more patients would have died if I had not been willing to explore the thorax; and if I had been



FIGS. 127 and 128.—Interlobar fissures and their relation to the ribs.

allowed to do exploratory thoracotomy sooner in Case 8, it is possible his life also might have been saved.

I must, however, put on record the following case, in which I opened the thorax after negative puncture and found no pus, the patient subsequently dying from pneumonia.

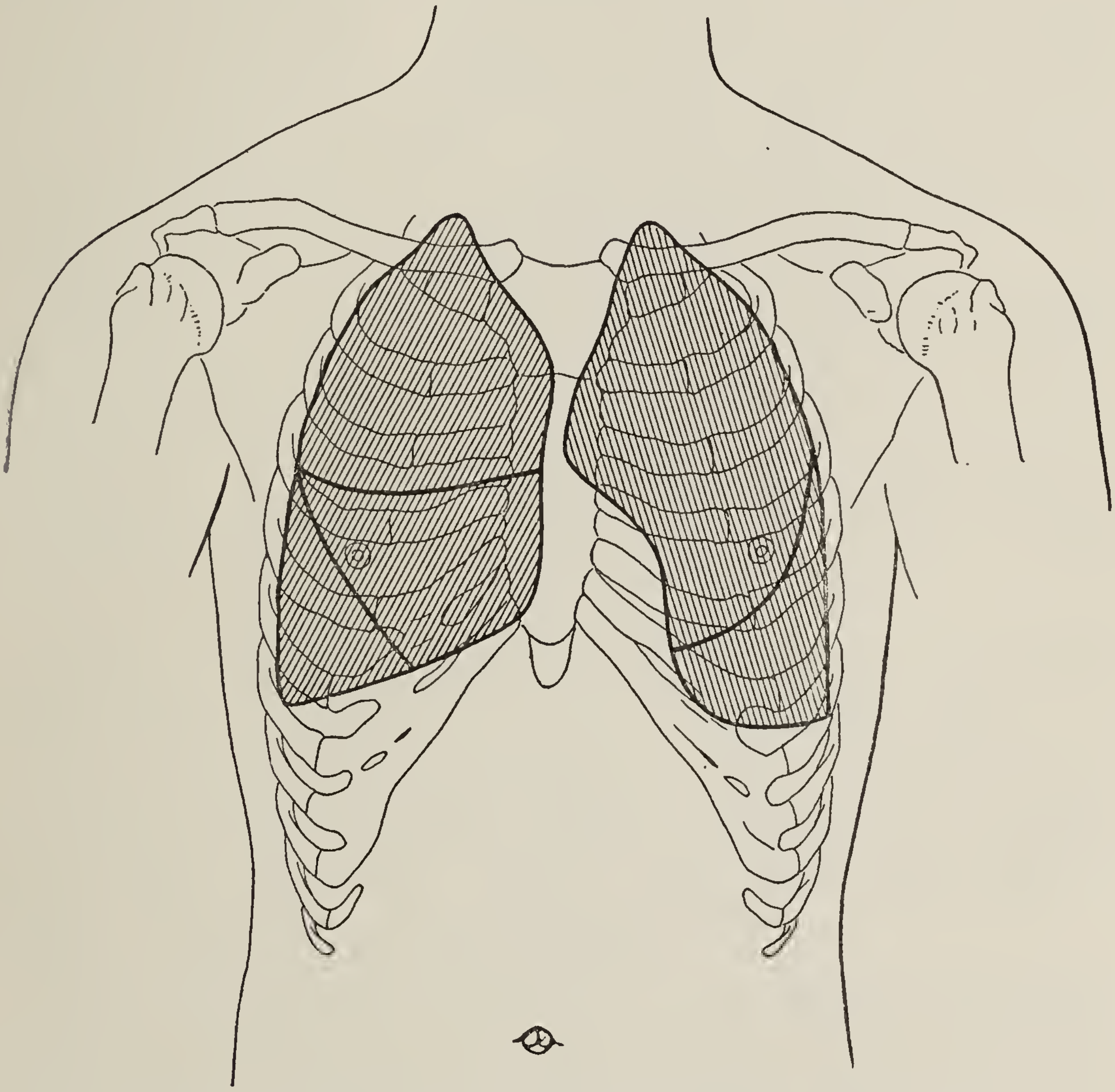


Fig. 129.—Fissures of the lungs viewed from the front.

Mildred, C., aged three years.

December 16, 1919. Admitted to the Episcopal Hospital, service of Dr. Carson, referred from Medical Dispensary by Dr. R. S. Hooker, with diagnosis of pneumonia. Has had a cold for three weeks, with cough and expectoration; not confined to bed. Is fretful and unable to breathe properly at night. On admission: Temperature, 101° F. (by rectum); anemic, rachitic child, with Harrison's groove and rachitic rosary pronounced. Respirations very short and rapid. Expansion of chest very asymmetrical.

Apex-beat of heart in midaxillary line about 6th interspace; marked pulsation over entire left chest. Rate rapid and sounds of good quality; no thrills or murmurs. Breath sounds rather harsh and many moist rales are heard over entire chest; no evidence of consolidation on percussion. Abdomen is distended, liver enlarged; no dulness in flanks. Extremities negative.

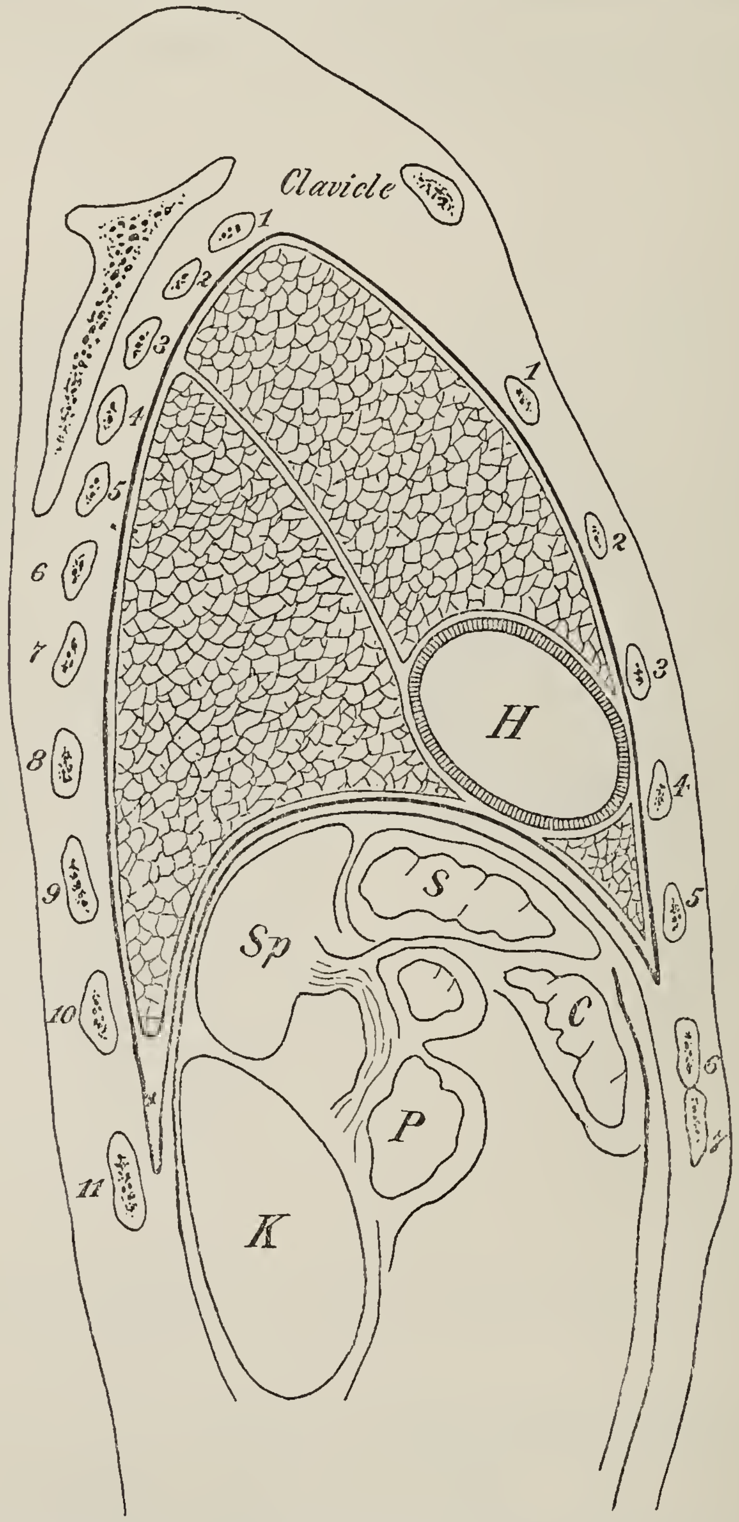


FIG. 130.—Sagittal section of thorax through middle of left clavicle.

December 17. Slight cough, no expectoration. Throat is clear. Not restless.

December 21. Improving. Temperature 98° to 100° F.

December 30. Temperature steady at 99° for last two days. Much improved. Sent home.

January 6, 1920. Brought to Medical Dispensary and again seen by Dr. Hooker. Since being home the cough has continued and the child's general condition has been poor. Diagnosis of left-sided empyema was made by Dr. Hooker from dulness and diminished breath sounds. Sent into Hospital and seen in receiving ward by Dr. Ashhurst. Puncture of left chest over area of impaired resonance is negative. Under local anesthesia 2 cm. of 10th left rib is resected, in midscapular line. Few adhesions between lung and diaphragm and between lobes of lung, but no pus found. Rubber tube inserted in expectation that pus will later rupture into drainage tract. Time of operation twenty minutes.

January 7. Condition poor. Lips cyanotic; respirations rapid and labored. No pus from tube. High-pitched, bronchial breathing at left apex.

January 8. Condition worse. Patient died at 5.15 P.M. Exploration of wound with finger after death could detect no adhesions between lobes and no evidence of empyema. Death from lobar pneumonia.

TECHNIQUE OF EXPLORATORY THORACOTOMY. Let me here repeat what I understand by exploratory thoracotomy:

The patient is given, about thirty minutes before the commencement of the operation, a hypodermic injection of morphin (0.010 gm.) and atropin (0.00045 gm.); these doses may be larger in robust adults and smaller in children.

The patient is placed *prone* on the table, is made comfortable with pillows and his head is turned away from the side on which operation is to be done. The arms are placed above the head. Care is necessary to see that the patient lies flat on his abdomen and thorax and is *not turned on the healthy side*, as this will hinder respiration.

The proposed skin incision, over the 8th or 9th rib, back of the angle of the scapula, is then infiltrated with the local anesthetic;[1] and the intercostal nerves above and below the rib

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[1] I prefer novocain, 1 to 400; but when this was not available, have used eucain 1 to 100; to every 30 c.c. of the solution is added one drop of adrenalin chloride solution (1 to 10,000). This quantity (30 c.c.) usually is sufficient.

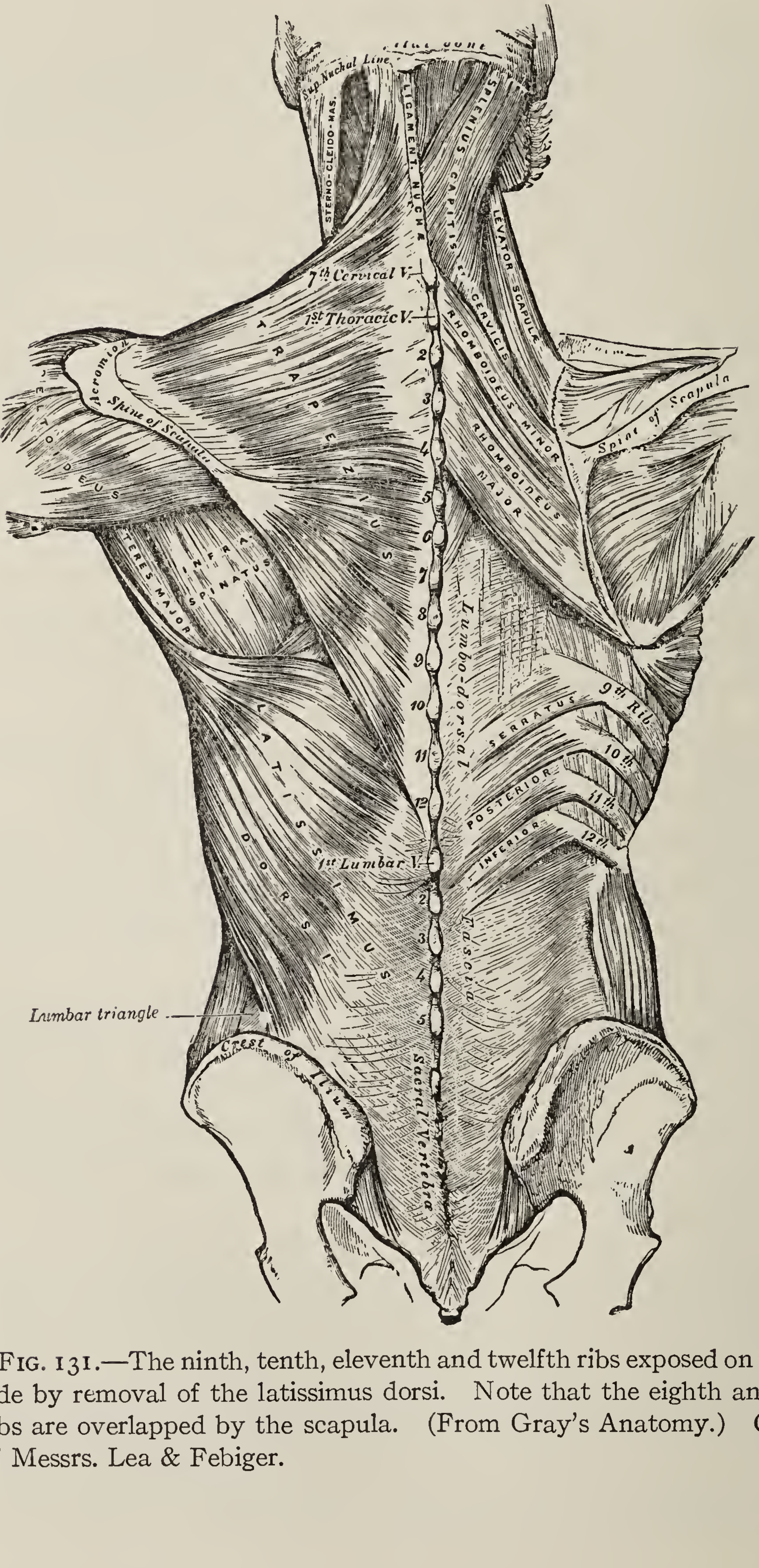

FIG. 131.—The ninth, tenth, eleventh and twelfth ribs exposed on the right side by removal of the latissimus dorsi. Note that the eighth and higher ribs are overlapped by the scapula. (From Gray's Anatomy.) Courtesy of Messrs. Lea & Febiger.

to be resected are blocked with an injection of about 2 c.c. each of the anesthetic fluid. These nerves are located by making the point of the needle impinge upon the rib above the nerve and then prodding the rib with the point of the needle until the lower border of the rib is found, when, after pushing the needle about 0.5 cm. further into the intercostal structures the injection is made. This should be at a point 8 to 10 cm. from the spinous processes. When the anesthetic has been properly injected the incision of the soft parts and the resection of the rib may be done absolutely painlessly. If, after the pleura has been opened and explored, more room is desired it is perfectly easy to resect the rib next above or below by (1) infiltrating the skin parallel to the spinal column upward or downward from the posterior end of the primary incision; (2) blocking a third intercostal nerve as already indicated; (3) raising the soft parts in a flap; and (4) resecting (subperiosteally) as much as is required of the next rib. Usually it is not necessary to divide the intercostal structures between the ribs resected, since when the ribs have been removed the intervening soft parts may be drawn aside by retractors. The length of rib to be resected depends on the size of the thorax and the extent of the intrathoracic exploration. As a rule, from 8 to 10 cm. is sufficient.

The pleura is opened between forceps, as the peritoneum is treated. Usually in cases of suspected empyema there is no collapse of the lung, which is adherent to the costal or diaphragmatic pleura. Even if the lung is not adherent it seldom collapses to less than half its bulk, when the patient is lying prone, and there is comparatively little respiratory disturbance. Wide opening of the pleura causes less dyspnea than a small and valve-like opening which favors the development of a tension pneumothorax.

After opening the pleura, and before conducting any exploration for an encysted empyema, the structures should be inspected. The lung may be so closely adherent to the diaphragm that it will be very difficult to recognize the line of junction. After ocular inspection, which may give a clue as to the first place to be searched for the abscess, the remainder of the pleural cavity should be walled off with hot moist gauze. Then with fingers, dissecting forceps or even knife and scissors (according to the density of the adhesions), the lung should be gently released. Usually it is well to clear the diaphragm first. If no pus is found here the packs are rearranged so as to protect the lower part of the pleural cavity, and a search is begun in the fissures of the lung. If the lung is not readily accessible its lower lobe is caught in two or three pairs of volsella forceps and maintained within reach. A single pair of volsella forceps is more apt to tear out and so damage the lung than are two or three simultaneously applied. Fixing the lung in the wound steadies the mediastinum, enables the diaphragm to resume its piston action, and thus ventilates the other lung. The lung is absolutely insensitive to these manipulations. Often it is not necessary to fix the lung in this way, and the surgeon proceeds at once to separate the lobes from each other. Usually where the adhesions are densest, pus will be found.

When pus is finally located, and has been evacuated, a rubber tube (with a lumen at least of 1 cm.) surrounded with loose iodoform gauze is placed in the abscess cavity, the isolating packs are removed, and the wound is closed in layers, but not too tightly, around the drainage tract—the pleura and intercostal structures in one layer, then the skin.

The frequency of encapsulated empyema I am sure is not recognized. In my own experience it has been present almost in one-third of the cases. It is true of course, that every empyema is, in a sense, encapsulated; in other words, that it does not involve the entire pleural cavity. Pleuritis is in a way analogous to peritonitis: only so long as the effused fluid is sero-pus is it unconfined by adhesions. By the time frank pus has formed, in every case, whether in the peritoneum or the pleura, adhesions set certain limits, large or circumscribed, to the cavity in which pus is found. The only "free pus" that can exist in a body cavity such as the pleura or peritoneum is pus which is suddenly effused in overwhelming quantities into previously normal pleura or peritoneum, by the rupture of a localized abscess (appendicular or pelvic abscess, empyema of the gall-bladder, abscess of the liver or spleen, encysted empyema, etc.). But what I understand by encapsulated empyema is either one which comes into contact with the costal pleura at no point (Cases 18, 24, 25, 26, 27, 36) or over so small an area that it is difficult if not impossible to locate pus until after the thorax has been opened (Cases 7, 8, 28, 29, 35) or until an empyema necessitatis has formed. It should be recognized also that there may be more than one distinctly encapsulated empyema in the same patient (Cases 16, 29 and 35), and that occasionally an hour-glass empyema may exist, the channel of communication being large (as in Cases 37 and 43) or so small that drainage of one loculus is ineffective in producing cure of the other (as in Case 38). It is on account of the occurrence of cases such as these that I urge that, if the condition of the patient permits, exploration should be adequate to ensure that no such encapsulated foci are overlooked. (I am not prepared, however, to adopt Lilienthal's "major thoracotomy" under general anesthesia, so long as I continue to find a medium thoracotomy under local anesthesia satisfactory.) In all cases with massive empyema, of course, removal of a large amount of the pus by aspiration should precede by one or two days the formal operation of thoracotomy and in no case where the fluid can be removed by aspiration need drainage by thoracotomy be regarded as an emergency operation (*third proposition*).

Let me revert now to the *fourth proposition* I have quoted at the beginning of this discussion, namely, "That drainage is best effected by making two openings, one at the lowest available point, and carrying a large drainage tube through the cavity from one opening to the other." It seems to me that the doctrine of through-and-through drainage merely follows as a corollary in cases where the primary opening has been made too high; in other words, that if the primary opening is made at the site of election, which I regard as the 9th or 10th (occasionally the 11th or 12th) ribs near their angles (Fig. 132), it is unnecessary to make a counteropening, as one is already at the "lowest available point." I repeat, that in cases of exploratory thoracotomy as well as in cases of massive empyema, I practise and recommend that the opening should be made at the site mentioned, even if the needle has shown pus in the 6th or 7th interspace. It is so nearly invariably the rule that the empyema cavity extends down to the diaphragm, and it is so easy after opening the pleura low down to drain an empyema which may extend already to within a few centimeters of the opening, by breaking through its limiting adhesions at their most dependent portion; and a higher opening unless supplemented by a counter-opening at the lowest available point gives rise to so very prolonged a convalescence, that I believe there are exceedingly few cases in which it is desirable to be limited in selecting the site for drainage by the location at which pus was found on puncture. Only once since I adopted this rule have I encountered an empyema situated so very high, and with the lower portion of the thorax so certainly intact, as to induce me to make a higher opening; in this patient (Case 28) the empyema appears to have formed between the lobes of the left lung and to have finally come into contact with the parietal pleura between the vertebral column and the scapula at the level of the spine of the latter bone; and yet the drainage tract at this site, though it was nearly at the lowest level of the empyema cavity, nevertheless required over four months for healing.

Resection of a rib I regard as important in all cases. Intercostal incisions alone do not afford adequate drainage, and frequently secondary operations are required. I have employed it only once (Case 4) in a baby six months old; a month later it was necessary to resect a rib to improve the drainage, but two months after the first operation the patient died with unhealed sinus, probably from sepsis arising in the inefficiently

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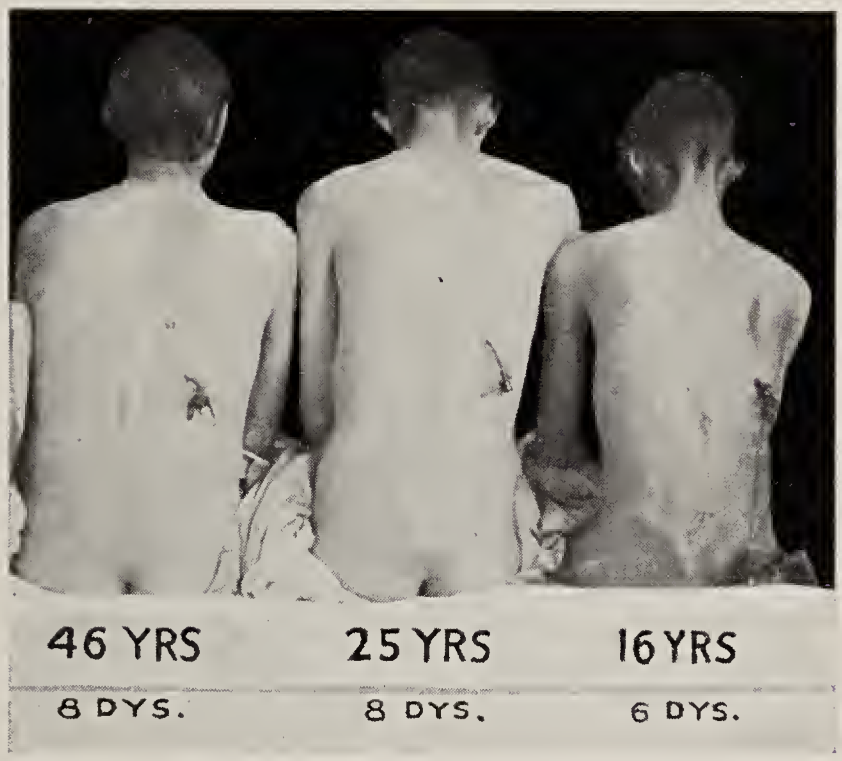

FIG. 132.—The site of election for draining an empyema; ninth, tenth or eleventh ribs posteriorly. The ages of the patients are given above, and the interval since operation below.

drained pleura. In another case which came later under my care (Case 13), intercostal incision was employed by another surgeon, but this healed upon an infected cavity, and the empyema being undrained externally ruptured into a bronchus; finally, the original operative sinus opened again, and eventually I had to resect the 8th and 9th ribs; definitive healing then occurred in eight weeks. I have never seen necrosis of the rib ends and cannot believe the possibility of such an occurrence is to be regarded as contra-indicating rib resection.

Next we come to the *fifth proposition*, "That drainage should be supplemented by washing out the cavity with mild antiseptic fluids. When the lung has expanded and discharge has nearly ceased the tube should be shortened . . . and gradually withdrawn." With the very gradual shortening and withdrawal of the tube I am in entire accord, but I do not see that anything is gained by irrigations in the ordinary case of empyema. I suspect that the main function they served in my father's day was to keep the lumen of the through-and-through drainage tube open, and that the fluid injected at one end of the tube almost immediately escaped by the other end, without making a very extensive journey into the empyema cavity. And I am quite sure that in some cases injudicious irrigations are productive of harm. It is quite evident, I believe, that death in two of my patients (Cases 30 and 31) some weeks after they passed from under my care is directly attributable to sepsis arising in the wound in the thoracic wall, not in the empyema cavity, and that this sepsis was produced by irrigation, and probably aided in Case 31 by too tight closure of the wound of the second operation.

That irrigations with Dakin's hypochlorite solution, by the Carrel technique, are of no value in any cases I am not prepared to admit. But I am quite thoroughly convinced that in the vast majority of cases of empyema they are quite unnecessary, and that except in cases of very long standing, with much thickened pleura and non-expansile lung, healing will occur quite as promptly, as pleasantly and as definitively by means of large and dependent drainage. I do not think it can be denied that in many patients of the class of cases just mentioned the irrigations with Dakin's solution will eat away the inflammatory lymph deposited on the visceral pleura and that the lung will thereafter expand sooner and more completely than if such irrigations had not been employed. Nor can it be denied that it is possible to sterilize an empyema cavity by this means, at least temporarily, and then to permit the thoracic wound to close upon the sterile pneumothorax; but I also know that the same event may take place simply as the result of efficient drainage without any irrigations, at any time (Cases 14 and 35). Speaking for myself, I may say that I have never employed the Carrel-Dakin treatment for empyema, and that I should be surprised to encounter a recent case in which I thought it indicated.

The *sixth proposition*, which has to do with unhealed empyema cavities, I am sure must undergo great modification in view of advances in surgery since it was formulated. I am convinced that unhealed empyema is infrequent compared to its incidence a generation ago, largely due to the earlier recognition and more efficient treatment of the acute cases. Indeed it is probably true that if any surgeon conscientiously follows up the treatment the occurrence of an unhealed case will be extremely rare. One patient (Case 6) on whom I operated in 1909 is known to have had his sinus still unhealed nine months after operation; but among the remainder of my patients the longest period to healing was seven months, and in not one of those who remained under my care was a second operation required. One patient (Case 31) who passed from my care two weeks after the operation, and from whom the tube was removed too early, was reoperated on later for an accumulation of pus, and eventually died of sepsis arising in the thoracic wound of the second operation. Another patient (Case 35) who was tuberculous and whose thoracic wound healed within seven weeks upon a pneumothorax, five months later had a little discharge from the sinus which healed again in three

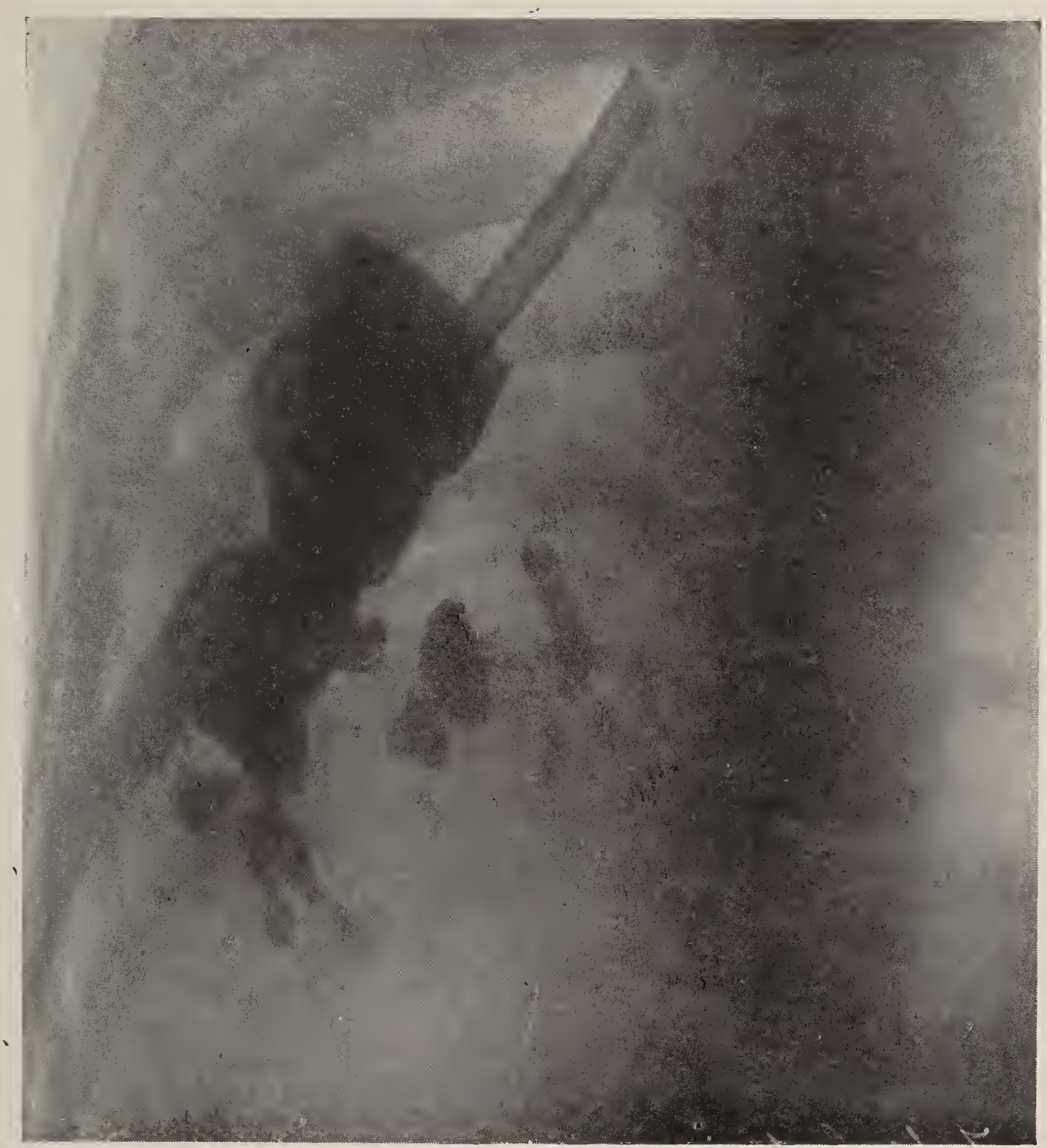

FIG. 133.—Rubber tube lost in empyema cavity for three months. Bismuth injections unavailing. Sinus healed permanently three weeks after removal of tube and Estlander operation.

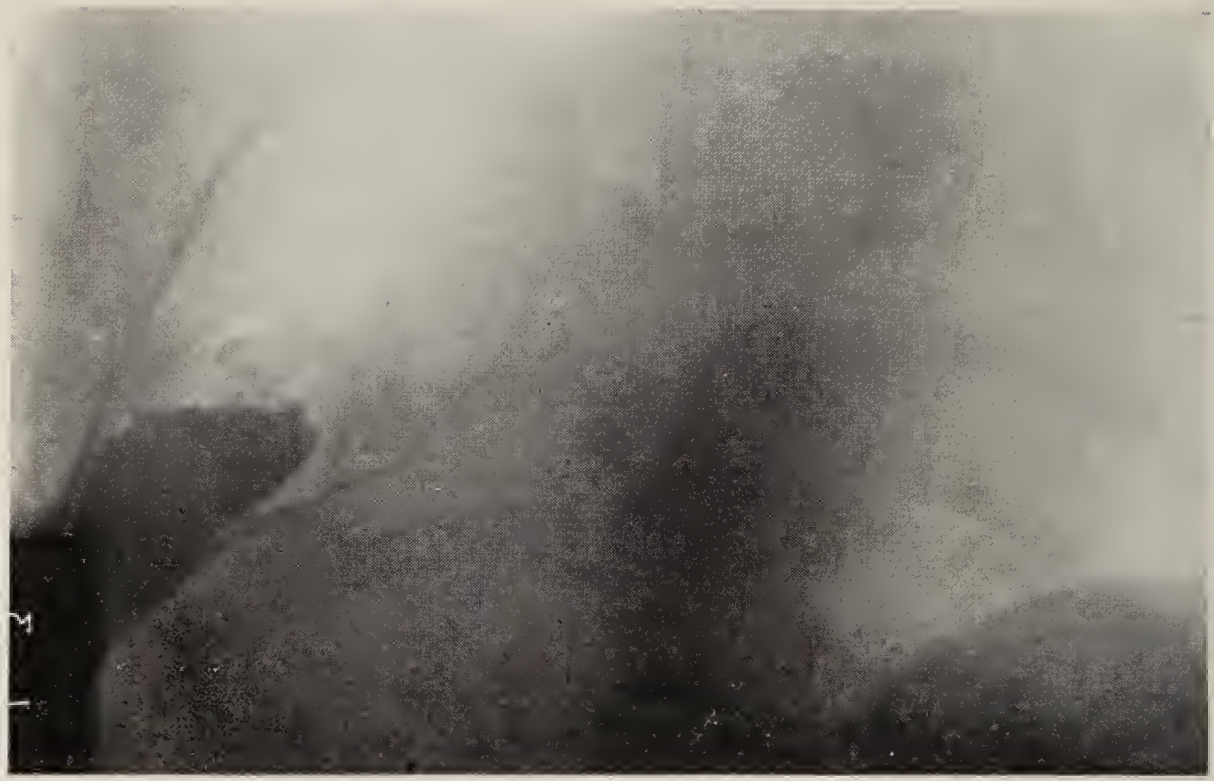

FIG. 134.—Empyema cavity, *L*, injected with bismuth paste, showing outlines of calcified pleura; ten months' duration. Died ten days after Estlander operation.

weeks; twenty-two months after my operation another empyema developed and was drained; seven months after this second operation the patient moved to Colorado, and since that time (September, 1919) he has had one slight reaccumulation which was opened and healed at the end of six weeks. He writes (March, 1920) that he is now apparently in perfect health.

I should like to say a good word for bismuth injections in the treatment of unhealed sinuses, as advocated by Ochsner. In one patient (Case 36) (the only one in whom I began to despair of seeing the sinus close spontaneously and in whom removal of the tube was followed on several occasions by rise of temperature), I had just about determined on reopening the wound (twelve weeks after operation) and injected it with 10 per cent. bismuth paste to ascertain the outlines of the cavity by skiagraphy; the next day the sinus was found healed, with the paste retained, and the wound remained healed when the patient was last seen five months later.

Unhealed empyema must be rare in Philadelphia, as I have had occasion to operate on not more than two patients.

In the *first patient*, John K., aged sixteen years, operation had been done by another surgeon April 19, 1911, for a left-sided empyema five weeks after the onset of pneumonia. On June 11 the tube was not found when the wound was dressed, and on June 17 the patient was discharged with unhealed sinus. Soon after this he came under my care in the Episcopal Hospital and was treated with bismuth injections. The sinus sometimes would close for a few days but always reopened. Finally a roentgen-ray picture was taken and showed the tube lying in the pleural cavity.

September 15, 1911 (three months after the first operation), under ether, I resected about 6 cm. each of the 7th, 8th and 9th ribs (the 8th, which had been excised at the former operation, was of cartilaginous consistency). The drainage tube and large masses of bismuth paste were removed and a fair amount of pus was evacuated. All the lymph adherent to the visceral pleura that could be detached was removed, all the adhesions of the lung to the parietal pleura were broken through and the cavity thoroughly wiped with iodoform gauze. One long and wide strip of iodoform gauze was left in for drainage.

In two weeks the wound was practically closed and was firmly healed by October 7, 1911, twenty-two days after operation. (March 1921. Has not had a day's illness since operation.)

In the *second patient*, James K., aged fifty-one years, there was a history of some severe injury to the chest twenty-eight years previously. He had had many attacks of "pleurisy" following occasional attacks of rheumatism.

May 24, 1913. Admitted to the Episcopal Hospital, Dr. Mutschler's service. About fifteen months previously he had developed a heavy cough followed by swelling in the left thorax, diagnosed by family physician as tumor. The family physician incised this swelling and quite a large quantity of pus was removed. The sinus healed up after a long time. There had been no discharge for the past four or five months. About ten days ago he began coughing again and the sinus opened and discharged pus. Has been having night-sweats. Does not spit any blood.

On admission: Sinus in left thorax between 6th and 7th ribs, in nipple line, from which small amount of pus is oozing. Two scars of healed sinuses. Temperature 101°. Large-framed man, thorax well developed, expansion limited on left, which is dull front and back below 4th rib, with distant breath sounds and harsh, crepitant rales.

May 26, 1913. Roentgen ray 6442—heart displaced to right by pus in left pleura up to 6th rib.

May 27. Operation by Dr. Mutschler (ether): 6th left rib excised in anterior axillary line and much pus discharged (pneumococci).

May 30. Temperature normal.

June 28. Still free discharge. Temperature hectic since last note, 98° to 101°. Roentgen ray 6640—heart still to right, some collapse of left lung.

July 25. Still same amount of discharge. Temperature, 98° to 100°.

August 1. Service taken over by Dr. Ashhurst. The patient walks about and does not complain of anything; general condition about same. Dressings always saturated overnight. Coughs a little.

August 8. Roentgen ray 7213 (bismuth) shows large cavity extending from 11th rib to upper thorax.

August 23. Discharge no less. Temperature, 98° to 100°.

August 29. Operation; ether. (Ashhurst.)

Patient prone. Incision 12 cm. long made over sinus of previous operation (over 6th left rib in anterior axillary line), and about 10 cm. each of the 6th, 7th, 8th and 9th ribs resected, from their angles forward. This uncovered very dense parietal pleura, which was found to be lined by a calcareous deposit about 5 mm. thick, as were also the exposed surfaces of the lung and diaphragm. The rigid walled cavity extended as high as the finger could reach through the wound. The calcareous lining was peeled off the parietal pleura, the diaphragm, and the lung, except at the apex of the cavity, where a few plates had to be left, as it was impossible to remove them without tearing the lung. The lung was found still to be bound down by dense pleura, beneath the calcareous deposit, but the condition of the patient did not warrant continuing the operation by decortication or discission of the pleura. The lung moved a little more in respiration than before the calcareous plates were removed, but it was evident that only by collapse of the soft parts could the cavity be obliterated; and though complete obliteration was not hoped for it was thought the marked reduction in size of the cavity would considerably reduce the discharge and promote the patient's comfort. Drainage was provided by iodoform gauze and a large rubber tube. The wound was closed in layers about the drainage.

August 31. Dressed, dressings soiled but not saturated, as before operation. No calcareous matter could be felt by finger introduced through wound.

September 6. Temperature fell after last dressing to normal and has not varied more than ½° since. Today rose to 100°. Seems irrational, no cough.

September 7. Restless, on gradual decline. Pulse weak, rapid (150). Temperature, 101°. Respiration, 34.

September 8. Died 6 A.M. Temperature having risen rapidly to 104°. No postmortem, but by opening wound calcareous matter found only around apex of cavity. Lung and diaphragm covered with gray lymph, phlegmonous in type.

It is a question in my mind whether in this latter patient the condition of his pleura did not date from his original injury twenty-eight years previously. Certainly it would be very unusual for such dense calcareous deposits to have occurred between the time of the two operations done in the Episcopal Hospital only three months apart; but the presence of a hemorrhagic pleural effusion many years ago, which remained unabsorbed, will account for the patient's repeated attacks of pleurisy as well as for the densely calcareous deposit. In this connection the appearance of the roentgen ray in Case 26 (Fig. 142) is suggestive that here also a cavity containing sterile fluid may have persisted for over four years, this patient having been under treatment in another hospital for supposed empyema, which could not be located, that length of time before she came under my care with recurrence of similar symptoms.

Conclusions. From this discussion it seems to me that the propositions laid down twenty-six years ago must be modified in the following particulars:

1. Cases of pleural effusion suspected of being purulent should be punctured (with hollow needle attached to air-tight syringe); and if the effusion is massive, most of it should be removed by aspiration one or two days before thoracotomy is undertaken.

2. If the fluid found on puncture is serous, or seropurulent, thoracotomy usually may be postponed, until frank pus has formed, as this delay will permit the formation of firmer adhesions and thus prevent complete collapse of the lung when the empyema is opened. Cures of such seropurulent effusions,

however, have so rarely occurred without final resort to thoracotomy that attempts to cure them by injection of antiseptic fluids into the unopened pleura are usually detrimental to the patient.

3. If in a case of suspected empyema the symptoms are urgent, but pus cannot be found by puncture, exploratory thoracotomy should be undertaken in an effort to locate and drain the pus.

4. The operation of thoracotomy for empyema should provide free and dependent drainage, secured by resection of a rib, usually the 9th, 10th, or 11th in front of its angle (Fig. 132). This operation may be done with perfect satisfaction to both patient and surgeon *under local anesthesia*, and in most cases this is preferable to a general anesthetic. (I have used local anesthesia with satisfaction in 27 patients, including one baby sixteen months of age; and have employed it in every one of my last 20 patients, some of them requiring rather extensive intrathoracic manipulations. It is interesting and perhaps significant to note that only two patients in the entire series (Cases 3 and 11) developed pneumonia of the opposite lung after operation; and that both these patients had taken ether.)

5. Postoperative irrigations are unnecessary, unless after several months the lung shows no tendency to expand; when the use of Dakin's fluid may prove beneficial. In selected cases (those with small cavity) injections of bismuth paste may procure closure of the sinus.

6. If the cavity cannot be made to heal by these means the surgeon should do a major thoracotomy, combined with decortication of the lung and discission of the pleura; and in some cases resection of a number of ribs to permit the chest wall to collapse in part and meet the expanding lung.

* * *

In closing, I may be permitted to give a summary of my own cases, few in number, perhaps, but studied with such attention as was possible. Among 43 patients there have been 9 deaths, a general mortality of 21.4 per cent. Among the first 9 of these patients there were 5 deaths, 3 of which were in babies less than a year of age. But if there are included only the last 34 patients operated on since adopting the system of wide and dependent drainage,[1] there were in this number only 4 deaths, a mortality of 12 per cent. Moreover, of these 4 fatal cases:

(*a*) In the first (Case 22) the child also had extensive gangrenous stomatitis (noma) and died from this.

(*b*) In the second (Case 26) the empyema (which was encapsulated against the pericardium) was not found at operation, and the patient died eight days after operation unrelieved.

(*c*) (*d*) In the third and fourth cases (Cases 30 and 31) death occurred some weeks after the patients passed from my care, and as the result not of their empyema, but from sepsis arising in the thoracic wound and brought on by irrigations.

From which facts I conclude that the mortality of empyema, if promptly and efficiently treated need not exceed 10 per cent.

The average time required for firm healing of the thoracic wound in 24 cases in which this is known, was just over nine weeks, the shortest time being three, and the longest thirty weeks. In those patients (18 in number) who remained under my personal supervision, the average time until firm healing was just over seven weeks; the shortest time being three weeks, and the longest twelve weeks.

In 5 patients (Cases 30, 31, 36, 39 and 40), two of whom (Nos. 36 and 39) were still under my personal care at the time, there was more or less interruption to the progress of wound healing, by pocketing of pus; and in 2 of my early cases (Nos. 4 and 5) where dependent drainage was not secured at the first operation, secondary operation was required some time after they passed from my care. Final closure of the wound was secured in all cases traced (32 out of 34 patients who recovered) without an Estlander or similar operation.

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[1] It is proper that I should give due credit to T. Turner Thomas, whose studies in 1913 turned my attention particularly toward the value of dependent drainage.

Certain cases in the subjoined list deserve more than passing attention:

Cases of encapsulated empyema, with limited contact with parietal pleura—Cases 7, 8, 9, 15, 16, 21, 28.

Cases of encapsulated empyema without any contact with parietal pleura—Cases 18, 24, 25, 26, 27, 36.

Cases of two distinctly encapsulated empyemata within same pleural cavity—Cases 16, 29, 35.

Cases of hour-glass empyema—Cases 37, 38.

Cases of empyema necessitatis, Cases 6, 17 (pulsating) 21, 38.

Cases of empyema as part of a general septicemia—Cases 14 (peritonitis and abscess), 24 (streptococci in blood), 33 (jaundiced), 34 (pericarditis, endocarditis), 38 (metastatic abscess and pyemia).

Cases of encapsulated empyema rupturing through a bronchus—Cases 8, 13.

Cases of tuberculous empyema—Cases 12, 17, 35.

Case in which drainage tube fell into empyema cavity—Case 16.

Case complicated by noma (gangrenous stomatitis)—Case 22.

Case complicated by independent pleural effusion on same side—Case 21.

The subjoined table gives the salient features in all the cases encountered, and, in accordance with the motto, "Ars medica tota est in observationibus," abstracts of the case histories are appended.

CASE HISTORIES

CASE 1.—Empyema, right. Recovery.

John C., five years.

September 22, 1906. Admitted to the Children's Hospital, Dr. Hutchinson's service.

Ill for six weeks with pneumonia at Atlantic City; then in Children's Hospital (Dr. Charles H. Weber's service) for the last three weeks. Empyema suspected, but Dr. Weber had four

ANALYSIS OF OPERATIONS FOR EMPYEMA.

No.	Name, age and date of operation.	Side affected.	Duration of illness before operation including a preceding pneumonia.	Metapneumonic or tuberculous; and culture.	Massive or encapsulated.	Operation and anesthetic.	Result.	Sinus healed.	Remarks.
1	John C. 5 yrs. Sept. 22, 1906	R.	6 weeks +	Metapneumonic pneumococci	Massive	8th rib; ether; pus, creamy, pale yellow, sour	Recovered	Under 10 mos.	
2	Joseph S. 5 months July 26, 1907	L.	3 weeks +	Metapneumonic diplococci	Massive	8th rib; ether; pus thick, inodorous	Died	..	Death in one week from enteritis.
3	Ed. W. 11½ months July 25, 1908	L.	2 weeks +	Metapneumonic diplococci and staphylococci	Massive	8th rib; ether; 100 c.c.; thick creamy pus	Died	..	Death in two weeks from pneumonia of other lung.
4	Kath. P. 6 months July 24, 1909	L.	4 weeks +	Metapneumonic pneumococci	Massive	Intercostal incision, 7th interspace; pus, 500 c.c.; ether; rib resected one mo. later	Died	..	Death in two mos. from sepsis.
5	Jos. T. 6 years July 24, 1909	L.	5 weeks +	Metapneumonic culture (?)	Massive	8th rib; ether; pus, 100 c.c.; second rib resected one mo. later	Recovered	Over 9 mos.	
6	Elias R. 3 years July 29, 1909	L.	5 weeks +	Metapneumonic culture (?)	Massive; "necessitatis"	6th rib; ether; 500 c.c. creamy pus	Recovered	Yes	Not traced.
7	Sam S. 5 years July 29, 1909	L.	11 days	Metapneumonic mixed (colon)	Encapsulated; parietal	8th rib; ether; pus thin and turbid; colon smell	Recovered	Yes	Not traced.
8	Wm. L. 50 years Sept. 14, 1912	R.	2 months +	Metapneumonic staphylococcus, diplococcus, bacilli, etc.	Encapsulated; interlobar; discharging thru bronchus	8th and 9th ribs; eucain; 30 c.c. extremely fetid pus	Died	*	Death in two days from sepsis.
9	Minnie Y. 59 years Dec. 25, 1912	L.	19 days	Metapneumonic pneumococcus	Encapsulated parietal	11th rib; cocain; semisolid pus	Died	*	Death in six days from bedsores, etc.
10	Mary O'B. 5 years Jan. 22, 1913	L.	2 weeks	Metapneumonic pneumococcus	Massive	9th rib; ether; 200 c.c. yellow, curdy pus	Recovered	*8 weeks	
11	James F. 7 years Feb. 14, 1913	L.	3½ weeks	Metapneumonic pneumococcus	Massive	11th rib; ether; 500 c.c. yellow, creamy and curdy pus	Recovered	3½ mos.	In good health seven years later.

12	Walter H. 15 years Aug. 26, 1913	L.	10 months	Tuberculous; smear: micrococci; culture: no growth	Massive	11th rib; ether; greenish pus	Recovered	*4 weeks	
13	Edna P. 7 years Aug. 27, 1913	L.	6 weeks	Metapneumonic diplococcus	Massive	Intercostal incision, 6th interspace; 8th and 9th ribs resected two months later; bloody pus	Recovered	4 months after intercostal incision *8 weeks after rib resection	
14	Margaret P. 12 years Nov. 1, 1914	L.	12 days	Followed peritonitis (appendicitis?); streptococcus	Massive	8th rib; gas; 150 c.c. pus; later, drainage of intraperitoneal pelvic abscess	Recovered	*10 weeks	Streptococcus grown from blood; also metastatic osteomyelitis of phalanx (staphylococci) and furunculosis
15	Hugh McI. 43 years Nov. 8, 1914	R.	4 weeks	Metapneumonic pneumococcus	Encapsulated parietal	9th rib; eucain	Recovered	Yes	In good health one year later.
16	Joseph R. 19 years Dec. 4, 1914	L.	3 weeks	Metapneumonic diplococci	Encapsulated; 2 parietal empyemata	8th rib; eucain	Recovered	?	Tube fell into cavity after operation.
17	Gerald L. 28 years Dec. 11, 1914	L.	3 weeks +	Tuberculous for seven years; streptococcus	Massive; *pulsating empyema necessitatis*	9th rib; eucain; prune-juice pus smelling of hydrogen sulphide	Recovered	?	Not traced.
18	Tony V. 2 years Nov. 23, 1915	R.	5 weeks	Metapneumonic streptococcus, staphylococcus	Encapsulated interlobar	7th rib; ether	Recovered	*10 weeks	In good health one year later.
19	Joseph F. 9 years Nov. 30, 1915	R.	17 days	Metapneumonic; mixed (colon)	Massive	9th rib; gas; 500 c.c. colon-smelling pus	Recovered	9 weeks	In good health nine months later.
20	Francis P. 11 years Dec. 3, 1915	R.	2½ weeks	Metapneumonic streptococci, diplococci, staphylococci	Massive	7th rib; ether; 200 c.c. pus; seven weeks later resection of 6th and 7th ribs; 150 c.c. thin fetid pus (streptococci only)	Recovered	5 months (3 mos. after second operation)	In good health one year later.
21	Joseph B. 34 years Dec. 10, 1915	L.	3 weeks +	Metapneumonic staphylococcus	Encapsulated; parietal *empyema necessitatis* with independent pleural effusion	11th rib; novocain; turbid fluid, 300 c.c.; intercostal incision, 7th interspace; thick yellow inodorous pus	Recovered	*7 weeks	In good health nine months later.
22	Edward S. 7 years Dec. 11, 1915	R.	3 weeks +	Metapneumonic staphylococcus	Massive	8th rib; gas	Died	*	Death one and a half days from noma of mouth.

* Indicates that the patient remained under author's care until final recovery or death.

ANALYSIS OF OPERATIONS FOR EMPYEMA—*continued.*

No.	Name, age and date of operation.	Side affected.	Duration of illness before operation including a preceding pneumonia.	Metapneumonic or tuberculous; and culture.	Massive or encapsulated.	Operation and anesthetic.	Result.	Sinus healed.	Remarks.
23	James C. 3 years Jan. 7, 1916	L.	3 weeks +	Metapneumonic pneumococcus	Massive	10th rib; ether; 250 c.c. creamy pus	Recovered	*9 weeks	Measles during convalescence; in good health 7 mos. later.
24	Philip G. 14 years Jan. 14, 1916	L.	4 weeks	Metapneumonic streptococcus	Encapsulated between lung and diaphragm	10th rib; gas; 500 c.c. pus	Recovered	7 months	Streptococcus from blood; in good health 4 yrs. later.
25	Wm. B. 30 years Jan. 14, 1916	R.	9 weeks +	Metapneumonic pneumococcus	Encapsulated interlobar	9th rib	Recovered	*3 weeks	Not traced.
26	Kate D. 38 years Aug. 5, 1916	L.	6 weeks +	Metapneumonic	Encapsulated; interlobar, against pericardium	7th and 8th ribs; novocain	Died	*	Death in eight days, unrelieved; pus not found at operation.
27	James McG. 30 years Nov. 17, 1916	L.	3 weeks	Metapneumonic; smear and culture negative	Encapsulated between lung and diaphragm	9th and 10th ribs; eucain	Recovered	*5 weeks	In good health three and a half years later.
28	Wm. B. 20 years Nov. 17, 1916	L.	4 weeks	Metapneumonic	Encapsulated; parietal and interlobar	4th and 5th ribs; eucain	Recovered	Over 4 months	In good health three and a half years later.
29	John L. 23 years Dec. 29, 1916	L.	3½ weeks	Metapneumonic pneumococcus	Encapsulated; two separate empyemata, both parietal	9th rib; eucain	Recovered	*7 weeks	In good health three years later.
30	Stanley K. 9 years Jan. 16, 1917	R.	2 weeks +	Metapneumonic; smear and culture negative	Massive	11th rib; eucain; reddish brown pus	Died		
31	Robert S. 7 years Jan. 18, 1917	L.	3 weeks +	Metapneumonic diplococcus, pneumococcus	Massive	11th rib; eucain; reddish brown pus	Died		
32	Whitman, L., 46 years Jan. 24, 1917	R.	3 weeks +	Metapneumonic diplobacillus	Massive	11th rib; eucain; 250 c.c. creamy inodorous pus	Recovered	6 weeks	In good health three years later.
33	Wm. B. 26 years Jan. 24, 1917	R.	2 weeks +	Metapneumonic pneumococcus	Massive	11th rib; eucain; 1000 c.c. curdy yellow pus	Recovered	Not traced	Desperately ill before operation (jaundiced).

34	Wm. D. 16 years Jan. 26, 1917	R.	3 weeks	Metapneumonic pneumococcus	Massive	11th rib; eucain; 500 c.c. creamy yellow pus	Recovered	*6 weeks	Desperately ill before operation (bilateral pleuritis, endocarditis and pericarditis); in good health three years later.
35	Edward H. 32 years Apr. 8, 1917	R.	10 weeks +	Tuberculous; metapneumonic diplococcus	Encapsulated; two distinct empyemata: (1) between lung and diaphragm; (2) parietal	9th rib; eucain	Recovered	*7 weeks	No lung symptoms three years later (lives in Colorado).
36	Oliver C. 21 years July 26, 1919	R.	3 weeks	Metapneumonic pneumococcus	Encapsulated between lung and diaphragm	10th rib; novocain; 150 c.c. creamy yellow pus	Recovered	*12 weeks	In good health eight months later.
37	Isadore M. 11 years Aug. 8, 1919	R.	19 days	Metapneumonic pneumococcus	Massive; "hourglass empyema"	10th rib; novocain; 1000 c.c. pus	Recovered	*3½ weeks	In good health seven months later.
38	Evelyn S. 19 years Nov. 18, 1919	R.	3 weeks	Metapneumonic streptococci	Massive; "hourglass empyema necessitatis"	9th rib; novocain; 250 c.c. yellow creamy, malodorous pus	Recovered	*8 weeks	Desperately ill before operation; metastatic abscess under L scapula during convalescence; in good health six mos. later.
39	Gertrude W. 4 years Dec. 6, 1919	R.	4 weeks	Metapneumonic diplococci and staphylococci	Massive	9th rib; novocain; 100 c.c. pus	Recovered	*6 weeks	In good health four months later.
40	Thomas W. 16 mos. Jan. 9, 1920	L.	3 weeks	Metapneumonic pneumococci	Massive	10th rib; novocain; thin serous pus	Recovered	14 weeks	In good health four months later.
41	Alice M. 29 years Feb. 20, 1920	R.	3 weeks	Metapneumonic pneumococci	Massive	11th rib; novocain; 500 c.c. creamy yellow pus	Recovered	*7½ weeks	In good health five months later.
42	Theresa D. 27 years Feb. 20, 1920	R.	17 days	Metapneumonic pneumococci	Massive	10th rib; novocain; 500 c.c. thin turbid flaky pus	Recovered	*7½ weeks	
43	James S. 34 years Apr. 9, 1920	L.	8 weeks	Metapneumonic pneumococci	Massive; "hourglass empyema"	10th rib; novocain; 2000 c.c. greenish yellow creamy pus	Recovered	9 weeks	In good health four months later.

* Indicates that the patient remained under author's care until final recovery or death.

dry taps. Last night pus was found (diagnosed as interlobar in position, by Dr. Weber).

September 22, 1906. Operation (Dr. Ashhurst); ether. Excised one inch right 8th rib, in midaxillary line. Pleura thickened (5 to 6 mm.); incised and incision dilated with hemostat. Pus oozed out with little force, then as child began to cry on coming out of ether (ammonia substituted for ether after opening pleura) pus was ejected with much force, creamy, pale yellow, sour. Pus appeared to extend from diaphragm up, not interlobar (only). Before operation respirations were 60, fell to 40 immediately after and to 22 to 24 after two hours. Pus gave a pure culture of pneumococci.

November 8, 1906. Tube out.

July 27, 1907. Seen at Country Branch of Children's Hospital, in excellent health; wound closed.

CASE 2.—Empyema, left. Died one week (enteritis).

Joseph S., five months.

July 26, 1907. Admitted to the Children's Hospital, Dr. E. B. Hodge's service.

Had been ill three weeks with pneumonia. Dr. Hand aspirated (7–25–07) about 50 c.c. of pus (diplococcus, biscuit-shaped).

July 26, 1907. Operation (Dr. Ashhurst); ether. Resected 2.5 cm. of 8th left rib, about 100 c.c. thick, inodorous pus. Time six minutes. Child very ill. Temperature normal after operation and did well for four or five days, then got severe enteritis, with high fever. Lungs clear, wound clean.

August 2, 1907. Died, one week after operation, from enteritis.

CASE 3. Empyema, left. Died two weeks (pneumonia of other lung).

Edward W., eleven and a half months.

July 25, 1908. Admitted to the Children's Hospital, Dr. E. B. Hodge's service.

Has been in Hospital about two weeks with pneumonia; temperature came to normal, but rose again on July 22 and July 23. Dr. Hand aspirated July 24 and got 25 c.c. pus.

Examination: No dyspnea lying down; lessened expansion, dulness, absence of breath sounds at left base. Mucous rales at apex.

July 25, 1908. Operation (Dr. Ashhurst); ether. Excised 2 cm. rib (about 8th) below angle of scapula, patient on back. About 100 c.c. thick, creamy pus, slight odor (staphylococci and few diplococci). Rubber tube.

July 28, 1908. Since operation almost no discharge of pus.

July 29, 1908. Temperature up, cyanosis, dull over lower part of upper lobe.

July 30, 1908. Dr. Hand suggested collection of pus.

July 31, 1908. Sinus explored with finger, only firm adhesions found, lung expands well. No pus by needle inserted in dull area. Bronchial breathing above empyema cavity.

August 4, 1908. Pneumonia in right lung discovered.

August 8, 1908. Died of pneumonia of right lung.

Case 4.—Empyema, left. Died two months (sepsis from pleura).

Katharine P., six months.

July 22, 1909. Admitted to the Children's Hospital, Dr. E. B. Hodge's service. Pneumonia four weeks ago. Aspirated (Dr. Ashhurst) and 100 c.c. pus (pneumococci) evacuated. Did well until July 24, when dyspnea returned.

July 24, 1909. Operation by Dr. Ashhurst (ether): Intercostal (7 to 8th) incision, left; about 500 c.c. of pus evacuated, and tube inserted.

July 27, 1909. Doing well.

August 2, 1909. Has had rapid respiration, 72 to 84; pulse, 160 to 170. Temperature 101° to 102° for last few days. Wound doing well. Eats well; bowels normal.

August 4, 1909. After taking ammonium carbonate, syr. of senega, respirations are only 60 to 70. Pulse, 140 to 150. Temperature, 99°. Still miserable.

August 14, 1909. Much improved.

August 19, 1909. No more improvement.

Later in August Dr. Hodge resected a rib to secure better drainage. In September the child was taken home; taking a milk mixture. Death toward end of September, two months after operation, perhaps from sepsis from the pleura, the wound not having healed.

Case 5.—Empyema, left. Recovery.

Joseph T., six years.

July 24, 1909. Admitted to the Children's Hospital, Dr. E. B. Hodge's service.

Had pneumonia five weeks ago. Aspirated ten days ago at home, and 1 liter of pus obtained. On admission the apex beat of heart is to right of sternum. Marked orthopnea; after aspiration of 100 c.c. no more pus would run.

Operation (Dr. Ashhurst) under ether, resection of 2 cm. of

8th rib in left anterior axillary line and evacuation of about 1 liter of pus and flocculent lymph.

July 27, 1909. Pulse still weak, no fever. Lying down for first time since operation. Bloody pus discharging.

July 29, 1909. Tube removed, only blood-stained serum discharged and smaller tube replaced.

August 3, 1909. Tube left out; nothing in sinus. Slight discharge.

September, 1909. Dr. Hodge resected another rib for better drainage. Doing well in end of month.

March 9, 1910. Readmitted and record shows only use of bismuth paste. Discharged April 23, 1910. "Discharge disappearing rapidly." Cannot be traced.

Case 6.—Empyema, left. Recovery.

Elias R., three years.

July 29, 1909. Admitted to the Children's Hospital, Dr. E. B. Hodge's service.

Pneumonia five weeks ago. Developed deformity of chest which family physician called "postpneumonic pigeon-breast." On admission had empyema necessitatis (left), apex-beat in second interspace right. Whole left thorax tense, with fluid between ribs and the skin and muscles. Aspirated by resident, large quantity of creamy pus.

July 29, 1909. Operation, ether. Resected 2 cm. of 6th or 7th rib in anterior axillary line and evacuated over 500 c.c. of less creamy pus.

July 30, 1909. Setting up and playing around bed.

August 4, 1909. Uneventful recovery after this date.

Case 7.—Empyema, left. Recovery.

Sam S., five years.

July 22, 1909. Admitted to the Children's Hospital, Dr. Hand's service.

Illness began July 18 with chill and fever. Has had temperature of 103° since. Pneumonia in left lung. Empyema suspected yesterday, but two punctures by Dr. Hand drew no pus. Transferred to Dr. Hodge's service July 29.

July 29, 1909. Today Dr. Ashhurst punctured twice in anterior axillary line, left, where auscultation and percussion indicated pus, but found nothing. Then punctured in 7th interspace in posterior axillary line, where was most tenderness and very slight edema, but no auscultatory signs, and drew very foul pus from considerable depth. Expectoration slight but very foul, like pulmonary abscess (mixed growth).

Operation by Dr. Ashhurst (ether). Resected 3 cm. of 8th left rib in posterior axillary line, without injuring pleura; suspected pulmonary abscess, but on incising pleura quantity of thin turbid pus (colon smell) squirted; later some coagula of lymph. Drainage tube.

July 30, 1909. Temperature normal, little discharge.

August 5, 1909. Temperature normal ever since operation.

August 25, 1909. Temperature up for one or two days, two weeks ago and again yesterday and today. Uneventful recovery thereafter.

Case 8.—Empyema, right, discharging through bronchus. Died third day (sepsis). (Pleural vomica.)

William L., fifty-two years.

July 26, 1912. Admitted to the Episcopal Hospital; under Dr. E. J. Morris's care until August 1, 1912, then on Dr. Robertson's service.

Family History. Father, grandmother and brother died of tuberculosis.

Personal History. Pertussis as child. Tobacco and beer to excess.

Present Illness. Began ten days ago as sudden sharp pain in right side, after lying on that side on grass for an evening. Pain gradually subsided after few days. Then got short of breath. Family physician gave medicine without effect. Walked to dispensary and sent to ward.

Examination on admission: Large full blooded, dyspneic; constant cough and expectoration. Breath very fetid. Right chest: flat below 4th rib with hyperresonance above. Left apex: impaired resonance. Rales and harsh breathing at right apex and below 4th rib. Absent breath sounds and fremitus. Expectorates 500 c.c. daily of very foul-smelling pus. Temperature 99° to 100°.

July 27, 1912. Constant cough. Cannot sleep, cannot speak above a whisper. White blood cells, 16,680. No tubercle bacilli found in sputum in five separate examinations; elastic tissue absent.

August 6, 1912. Seen by Dr. Eves for aphonia; examination negative except for acute laryngitis.

August 18, 1912. Growing weaker. Temperature ranges from 99° to 102° F.

September 3, 1912. Aspiration negative on right, below angle of scapula. (Dr. Robertson.) Still septic.

September 7, 1912. Tapped by Dr. Robertson (negative) below angle of right scapula. Oxygen introduced into pleura until dyspnea was produced and entire area of dulness became tympanitic. Dyspnea soon disappeared and patient became comfortable.

September 9, 1912. Dulness as before over right chest.

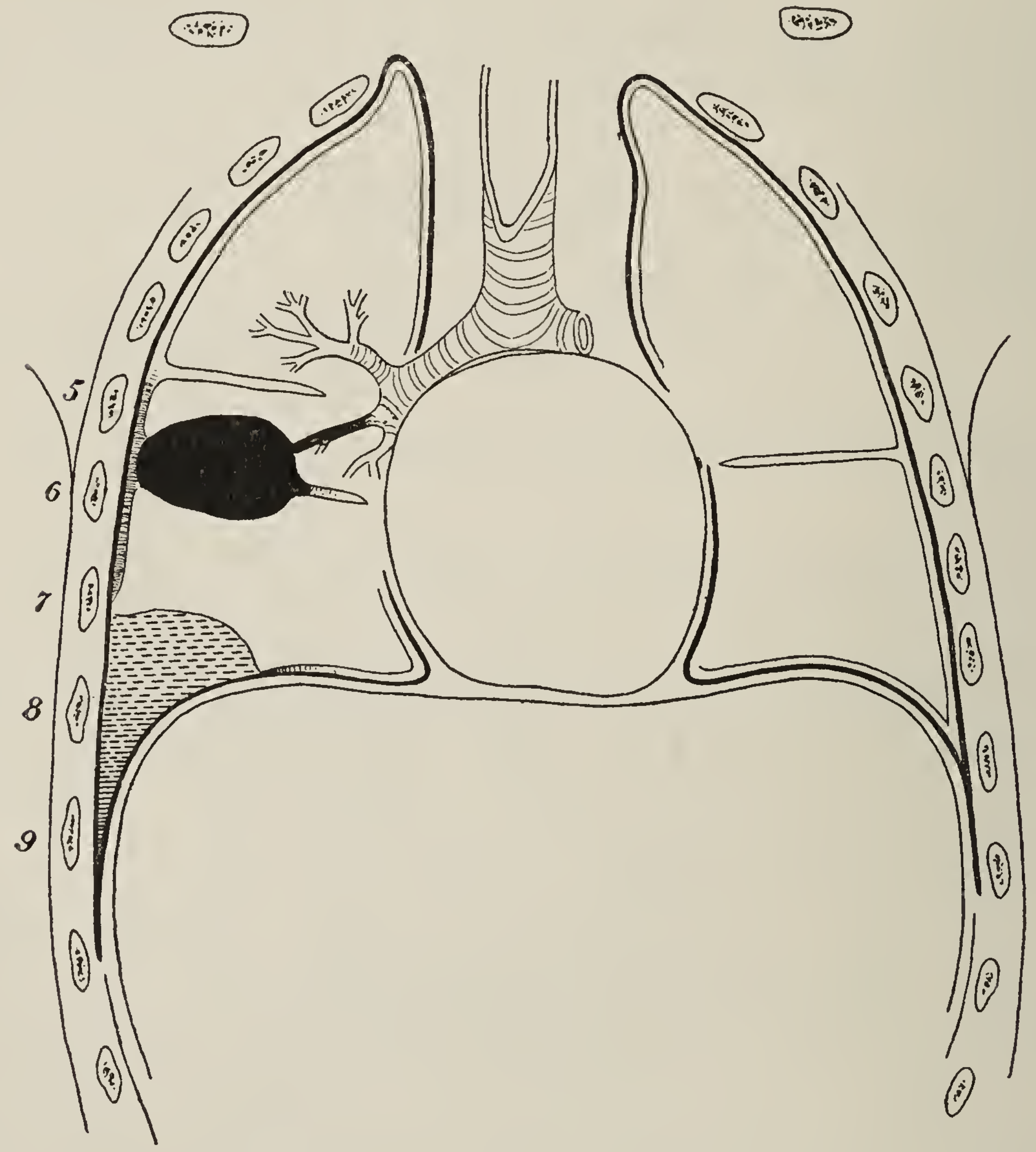

Fig. 135.—Case VIII. Interlobar empyema ruptured into a bronchus (pleural vomica). Note pleural effusion in costophrenic sinus.

September 10, 1912. Oxygen again introduced by Dr. Robertson. Patient nearly died; was unconscious for fifteen minutes; pulse 150.

September 11, 1912. Improved, dyspnea disappeared. Temperature 99° to 103° F.

September 13, 1912. Transferred to Dr. Frazier's surgical service. Seen by Dr. Ashhurst, who thought it to be an encapsulated empyema rupturing through bronchus. Patient is very dyspneic and coughs frequently until expectoration of very foul greenish material. Pulse fairly strong (92 to 96). Respiration 28 to 32. Temperature 100° and no pain. Whole right chest is flat from middle of scapula down; breath sounds weak, many crackling rales. Breath sounds amphoric below angle of scapula for distance of two ribs. Repeated punctures have located no pus.

September 14, 1912. Operation by Dr. Ashhurst (eucain 2 per cent. in intercostal nerves of 8th and 9th ribs and by infiltration also). Incision in 8th interspace posterior axillary line, patient lying on face. Excised 5 cm. of 9th rib and 6 cm. of 8th rib. Ligated both intercostals front and back. Patient complained of pain *in abdomen.* This exposed diaphragm. Bloody serum evacuated on opening pleural cavity; lung stripped easily from ribs except beneath scapula (6th to 8th ribs, in front of angles). Here it was densely adherent (culture of serous fluid; staphylococci, diplococci, bacilli). Packed remainder of pleural space. Lung densely adherent to dome of diaphragm and to posterior parietal pleura beneath scapula. Burrowed with finger among these adhesions beneath scapula, and ruptured into abscess, which discharged with a gush 30 c.c. or more of same pus as is expectorated. (Aromatic ammonia to stifle fetid stench.) This cavity was beneath 6th and 7th ribs in posterior axillary line, about 7 by 5 cm., though finger could not reach upper limits. Inserted rubber tube (1.25 cm. in diameter) and iodoform gauze around tube. Time one-half hour. Patient asked for his supper as soon as he was turned over on his back, and was given it on return to ward.

September 15, 1912. Very dyspneic, pulse very rapid and weak. Tube to lung drains large amount of greenish pus. Dressed wound.

September 16, 1912. Died 4.30 P.M.

CASE 9.—Empyema (encysted) left. Died six days (sepsis).

Minnie Y., fifty-nine years.

December 10, 1912. Admitted to Episcopal Hospital, Dr. Piersol's service. Ill for four days, with pain in left chest. Examination showed pneumonia of left lower lobe.

December 15, 1912. Temperature fell by crisis (ninth day).

December 20, 1912. Empyema suspected; dulness persists; impaired fremitus. Very ill.

December 24, 1912. Temperature suddenly rose to 104.3°. More toxic. Tapped, thick greenish pus from left pleura, base of lung. (Culture: pneumococcus.)

December 25, 1912. Transferred to Dr. Frazier's surgical service. Patient appears to be insane. Very emaciated and weak; bed-sore, etc.

Operation (Dr. Ashhurst). Cocain 2 per cent. Patient prone. Injected intercostal nerves at and below 11th rib at angle. Then waited five minutes. Skin not anesthetized yet, so infiltrated it (15 minims of 2 per cent. cocain. Same amount had been used for each of two nerves). Then entire operation (resection of 1½ inches of 11th rib) was done absolutely painlessly. After removing rib, found pleura unruptured. Opened this (as peritoneum) with scalpel and found lung lightly adherent, and showing no tendency to collapse. Packed pleural cavity off above with one strip of gauze, then burrowed in region of densest adhesions (just anterior to opening) and thick (semi-solid) pus welled up with each respiration. Inserted ½ inch rubber tube (about 2½ inches inside thorax), packed it around with iodoform gauze; withdrew isolating gauze; dressed wound, and turned her on her back.

December 27, 1912. Doing well. Temperature fell to normal after operation and remained there.

December 29, 1912. Temperature, 96°.

December 30, 1912. Temperature, 103°. Lungs negative. Drains well. Bed-sore slough removed.

December 31. Died from exhaustion and sepsis from bed-sore.

CASE 10.—Empyema, left. Recovery.

Mary O'B., five years.

January 10, 1913. Admitted to the Episcopal Hospital, Dr. Piersol's service. Ill three days with pneumonia of left lower lobe. Temperature reached normal by lysis on January 17, 1913.

January 20, 1913. Temperature 101° to 104°. Aspiratio in 9th left interspace gave a large amount of pus (Diplococcus, pneumococcus).

January 22, 1913. Transferred to surgical service. Temperature still high.

Operation (Dr. Ashhurst), ether. Patient prone. Resected 3 cm. of 9th rib, in front of its angle; gush of yellow curdy pus (about 250 c.c.). Drain: Rubber tube.

February 18, 1913. Tube discontinued.

March 10, 1913. Out of bed.

March 21, 1913. Discharged. Wound healed.

September 28, 1916. Social Service Department reports: has moved and cannot be found.

Case 11.—Empyema, left. Recovered.

James F., seven years.

February 10, 1913. Admitted to Episcopal Hospital, Dr. Piersol's service. Ill three weeks at home, feverish and coughing. Effusion in left lower chest. Needle gave thick, greenish pus. Culture: pure pneumococci. Temperature, 102°. Delirious.

February 12. Temperature normal. Flat all over left chest posteriorly.

February 14. Operation by Dr. Ashhurst (ether). Patient prone. Excised 3 cm. of 11th rib at angle. Gush of pus when periosteum was torn through before fragment of rib was excised. No hemostats required. About 500 c.c. of yellow, inodorous, creamy pus, with large amounts of lymph. Lung not much bound down by adhesions and expanded well after removal of pus. Drain tube, 1 cm. lumen, inserted about 5 cm., so as to be above level of diaphragm when this rose.

February 17. Pneumonia at right apex. Delirious, very weak. Temperature since operation 99° to 102° F.

February 21. Temperature normal.

February 27. Bed-sore over sacrum.

March 11. Slowly improving.

March 21. Tube replaced.

April 3. Bed-sore healed.

April 11. Tube removed.

April 20. Tube replaced.

April 28. Tube removed. Out of bed.

May 10. Discharged. Sinus gives slight discharge.

September 13, 1916. Healed three or four weeks after leaving hospital. No deformity; rib has reformed. Impaired resonance and a little distant breath sounds. Well and hearty.

April 28, 1920. Mother reports he is in good health.

Case 12.—Empyema, left. Recovered.

Walter H., fifteen years.

August 21, 1913. Admitted to Episcopal Hospital (Dr. Frazier's service).

Family History. No tuberculosis.

Personal History. Pneumonia at five years; well since. Schoolboy.

Present Illness. In October, 1912, noticed some swelling on left side, dull pain. Broke open in one month and discharged a little pus. Dressed twice daily since. Slight cough, frequent night-sweats. Very little expectoration.

Examination: Right apex, impaired resonance. Left is fair. Right apex bronchial breathing and pectoriloquy. After each five or six beats of heart a very loud friction rub is heard, evidently pericardial, and systolic in time. From 6th rib to base on left is dull front and back. Scars of two healed sinuses, anteriorly in 7th interspace. Sinus below angle of scapula discharges whitish, inodorous pus. Probe enters four inches and detects bare carious rib.

August 23. White blood cells, 18,320.

August 26. Operation (Dr. Ashhurst), eucain, 2 per cent.; 1½ inches of 10th left rib removed; great amount of greenish pus exuded (smear: few micrococci; culture: negative). Tube.

August 28. Not much pus.

September 6. Discharge less, irrigated daily.

September 8. Blows Wolfe bottles.

September 12. Better expansion on left.

September 20. No discharge. Sent home. Temperature never above 99° after operation.

September 28, 1916. Three years after operation reported to have left the city.

Case 13.—Empyema, left. Recovered.

Edna P., seven years.

June 25, 1913. Admitted to Episcopal Hospital, Dr. Mutschler's service. Had been ill for six weeks. Acute onset with meningeal symptoms. In bed since with pain in left side. Weakness and cough and yellowish expectoration. Whole left chest full of fluid.

June 27. Operation by Dr. Mutschler. Intercostal incision 6th to 7th ribs; left; large amount pus evacuated.

July 1. Improved. Temperature irregular but not high.

July 20. Only small sinus remains.

July 29. Temperature rising.

August 1. (Service transferred to Dr. Ashhurst.) Temperature 103°. Signs of fluid; two taps negative. Old sinus entirely healed.

August 2. White blood cells, 26,000.

August 5. Temperature 100° to 102°. Roentgen ray.

August 10. Coughed up some pus; sent to laboratory. Negative for tubercle bacilli; diplococcus predominated.

August 13. Sinus ruptured during night. Temperature lower.

August 19 to August 24. No improvement. Patient feels ill. Temperature up.

August 25. Better; thick yellowish expectoration. Roentgen ray after bismuth injection shows cavity extending from 4th to 11th ribs.

August 26. Temperature normal.

August 27. Operation, ether (Dr. Ashhurst). Patient prone. Excised 3 cm. of 9th and 8th ribs, near angle, opening cavity in pleura (no communication with bronchi apparent); removed bismuth; broke up some adhesions. Drain tube and iodoform gauze. Large amount of bloody pus removed.

September 18. Tube removed.

October 22. Entirely healed; out of bed.

October 27. Discharged.

September 6, 1916. Very slight scoliosis convex to left. (Left ribs were resected.) Ribs reformed. No disability. Attends State Dispensary for Tuberculosis.

February 21, 1920. Nearly seven years since operation, patient now fourteen years of age. Grandfather, living in same house, died of phthisis soon after her return home from hospital. Examination shows expansion of chest limited at both apices, but the lungs are normal. There is no increase in the slight left convex scoliosis noted three and a half years ago, and no disability.

Case 14.—Empyema, left (pleuroperitonitis). Recovered.

Margaret P., twelve years.

October 27, 1914. Admitted to the Episcopal Hospital on Dr. Fussell's service. Ambulance doctor (R. J. Sprowl) found temperature 105°, pulse 140 to 150; his diagnosis was typhoid fever. Had been ailing off and on for three weeks before onset of acute illness, but attended school daily. One week before admission had sudden violent pain in lower left abdomen. Began vomiting and has been vomiting ever since. Castor oil not effectual. Bowels not open for one week. No cough. Pains her to be moved and to take deep breath. Pains to urinate or make any motion with abdominal muscles. Examination on admission by Dr. J. W. Moore, receiving ward physician: Temperature 103°, pulse 136 to 120. Well-developed and well-nourished, but anemic. Lips red, face pale. Breathing rapid, but rather full respiration. No edema or cyanosis. Not apathetic. Teeth good. Tonsils very large and red and pharynx red. White blood cells, 30,000. Polynuclears, 75 per cent. Hemoglobin, 75 per cent. Red blood cells, 4,220,000.

Chest: Expansion good and equal on both sides. No thrills or frictions. Tactile fremitus normal everywhere. Whole lung resonant to percussion except an area the size of a quarter dollar under angle right scapula, which is dull, and an area in right midclavicular line 4th rib (anteriorly), which seems hyperresonant, almost tympanitic. Breath sounds vesicular *all over*, including dull area, but here is heard an occasional crackling rale. No bronchophony or whispered pectoriloquy.

Heart: Negative.

Abdomen: Very rigid and tender all over, especially in epigastrium, right and left iliac fossæ. Not scaphoid nor markedly distended. Spleen and liver not palpable because of rigidity, but spleen not enlarged to percussion and liver dulness present. Abdomen resonant throughout; no dulness in flanks. Active peristalsis all over abdomen.

Rectum: No masses or areas of exquisite tenderness.

October 28. Examination by Dr. Fussell:

Patient looks ill. Complains of pain in abdomen when moved. There is a red flush on right forehead. Constantly makes outcries because she states she had pain in her stomach. Slight crusted scar on lower right lip, may or may not be herpes. Slight glandular enlargement in both axillæ and both groins. Breathing rapid and rather quick. No dulness on left side in front or laterally. Dulness on right beginning at 5th rib in midaxilla and merging into liver dulness. No dulness on right side behind. No blowing breathing posteriorly on either side. During examination dulness in right axilla disappears. Liver dulness begins at 4th rib, parasternal line, and extends almost to edge of the ribs. Splenic dulness is apparently increased. In respiration the abdominal wall is absolutely fixed. On palpation there is no mass nor any particularly tender point. There is a great deal of abdominal tenderness in addition to the ridigity. Halfway between xiphoid and umbilicus the respiratory murmurs are curiously well heard. There is no sign of peristalsis. (Treatment just after admission is not recorded.) Rectum is ballooned out, but there is no mass.

Transferred to Dr. Ashhurst's service (day after admission). Temperature, 103°. Pulse, 120 to 130. Respiration, 38 to 40. White blood cells, 31,000. Eyes bright, cheeks red, rapid costal breathing. Abdomen very rigid all over, a little distended below umbilicus. Three doctors had examined her by rectum and felt no mass. Dr. Ashhurst did not examine chest or rectum; he thought it a surgical case (peritonitis), but that child was too sick for operation. Transferred to surgical ward and enteroclysis given; *nothing* by mouth (still vomiting frequently and has been having water in medical ward, but no enteroclysis) and continued head-high posture. Also camphor and digitalis hypodermically (not given in medical ward).

October 29. Got out of bed last night and managed to get a little water (delirious). White blood cells, 23,840. Polynuclears, 92 per cent. (Nothing by mouth, continued enteroclysis. Fowler position.) Abdomen distended, tympantic to percussion everywhere, save in right iliac fossa, where a dull area can be made out. Right side more rigid than left. Has vomited only twice. Nosebleed at 3.30 A.M.

October 30. Much weaker and more restless. Abdomen not so distended but board-like rigidity over entire abdomen. Does not vomit, has not passed any flatus; peristalsis can be heard in upper abdomen; complains of great thirst. Still nothing by mouth and enteroclysis. Temperature, 101° to 103°. Pulse, 120. Respiration, 26 to 30. Given digitalis and strychnin. No sign of localization of peritonitis.

October 31. (Saturday.) Decidedly better. Upper abdomen just a little softer. Suspicious boggy feel by rectum (first examination by Dr. Ashhurst). Respiration still very rapid. Temperature, 99°. Pulse, 110 to 115. By noon, hot water given by mouth. By afternoon (third visit by Dr. Ashhurst this day) feels almost convalescent she says. Complained of pain "around heart" and left lower chest at A.M. visit. P.M. Temperature, 101°.

November 1. (Sunday.) Temperature, 101° (steady). Pulse, 108 to 120. Respiration, 32 to 40. Respirations still very rapid. Left cheek flushed. Alæ nasi play violently. Abdomen rather rigid, no dulness; rectal examination, negative. Chest negative anteriorly. Dr. Ashhurst determined on thorough chest examination (his first) because of failure of abdominal abscess to appear. No evidence of subphrenic abscess (Hoover's sign). Right lung negative. Left dull from angle of scapula down, posteriorly and in axillary line, not anteriorly. Tender on pressure in intercostal spaces here. *Perhaps* slight bulging of these spaces. Breath sounds distant. *Needle* inserted in 7th interspace below angle of scapula drew pus (diplococci in chains). Dr. Fussell came in consultation; said subphrenic abscess, *no* evidence of empyema. Dr. Ashhurst maintained it was *empyema thoracis.*

Operation by Dr. Ashhurst (gas) 2.30 P.M. (Resected 8th rib in anterior anxillary line.) 150 c.c. (streptococci) pus from pleura. Tube. Liquids by mouth. At 10 P.M. abdomen less tender, lungs edematous. Heart holding out well. ℞—morphin and atropin. Temperature, 104°. Pulse, 120 to 140. Respiration, 40 to 48.

November 2. Profuse discharge of pus from pleura; heart displaced to right. Abdomen softer, normal bowel movement (first since enema October 29). White blood cells, 47,760. Polynuclear, 89 per cent.

November 3. Temperature falling. Continue morphin, atropin and stimulation. Three normal bowel movements (no purge).

November 4. Better, abdomen softer, no bowel movement.

November 5. Temperature gradually falling. Pulse, 120 to 130. Respiration, 30 to 38. Bowels open daily from now on. Examination by Dr. Fussell: Labored breathing. Heart: Right border one and a half inches to right of sternum, sounds are very rapid and feeble. Right chest anteriorly and posteriorly many rales. Left chest: pneumothorax. Abdomen is tender, especially over McBurney's point.

November 6. Examination by Dr. Ashhurst. No rigidity of abdomen, no tenderness to a reasonable degree of pressure over appendix or any other part of abdomen. To all intents and purposes the abdomen is normal. Respirations are thoracic. Fair expansion on both sides of upper chest; below nipples expansion better on right than left. No breath sounds heard anteriorly on left; normal on right. Right chest posteriorly is normal; on left posteriorly, pneumothorax.

November 7. Herpes on lip and tongue.

November 8. Improved.

November 10. Profuse discharge from chest still.

November 14. Fluid suspected at right base, no puncture.

November 16. Tube shortened.

November 22. Tube removed. Heart in normal position. Left thumb opened for infection (pure staphylococcus).

December 5. Out of bed. Thumb badly infected. Furunculosis of back.

December 11. Convalescent for two weeks and in chair now and chest normal. Excessive thinness.

December 14. Chest healed.

December 17. Gained three pounds in last three days.

December 23. Severe cough. Examination by Dr. Piersol: Compensatory emphysema of upper and middle right lobes; slight fluid or thickened pleura at right base from 7th dorsal spine down. Consolidation or compression of upper left lobe; below effusion and air.

December 24. Feels fine. Examination by Dr. Ashhurst; dulness at right base, breath sounds distant, consolidation; at left base sounds diminished, pneumothorax. Abdomen negative.

December 25. This afternoon had attack of acute pain in abdomen; vomited once. Temperature, 100°. Abdomen tender throughout and some rigidity.

December 26. Temperature, 99° to 101°. Right lower quadrant rigid. Liquid diet.

December 30. Sequestrum removed from thumb.

December 31. Chest unchanged. Aspiration of right; no fluid.

January 3, 1915. Temperature, 99° to 101°. Pulse, 104 to 120. Respiration, 28. Vomited this A.M.

January 4. No better. Slight pneumothorax anteriorly on left; empyema scar solid.

January 5, 1915. Examination by Dr. Frazier (rectal and abdominal), who found no abscess. Resident physician (Sprowl) could not feel anything by rectum. Dr. Ashhurst examined patient again and found all abdomen below navel was rigid and tender and dull, and in rectum a distinct bulging abscess. White blood cells 26,000 for last week. Polynuclears five days ago 82 per cent., yesterday 72 per cent. *Diagnosis:* Pelvic abscess from appendix, relighted by Christmas dinner.

Operation: Gas. 11.30 A.M. Incision right paramedian two inches, suprapubic. Transversalis fascia and peritoneum matted together and dense; at once on opening peritoneum, white, creamy pus welled out, about 50 c.c. in all. Finger inserted; felt intestines matted together by adhesions; and burrowed to Douglas's pouch. Drain: rubber tube, and iodoform gauze. No sutures. Time ten minutes. (Culture: Streptococcus.)

January 6. Temperature normal, free discharge of pus; much better.

January 10. Bloody purulent expectoration. Sitting in chair.

January 13. Tube removed from abdomen.

January 15. Weight 54½ pounds.

January 17. Slight discharge. Gaining weight.

January 23. Went home, weight 59 pounds. Sinus closed.

October 4, 1915. Scar two inches. No hernia. No disability. Appendix has never been removed.

April, 1920. Patient cannot be traced. Family report she was in good health a year ago, when she was employed as a professional dancer.

CASE 15.—Empyema, right. Recovered.

Hugh McI., forty-three years.

November 7, 1914. Admitted to the Episcopal Hospital to Dr. Frazier's service.

Ailing all summer. Pneumonia four weeks ago, crisis three weeks ago, but did not get well; had fever, cough and marked expectoration. In bed all the time. Bed-sore beginning. Temperature 101° F.; right lung from middle down gives absent breath sounds and fremitus, and is flat. Aspiration gives pus in 7th interspace posteriorly (pneumococcus).

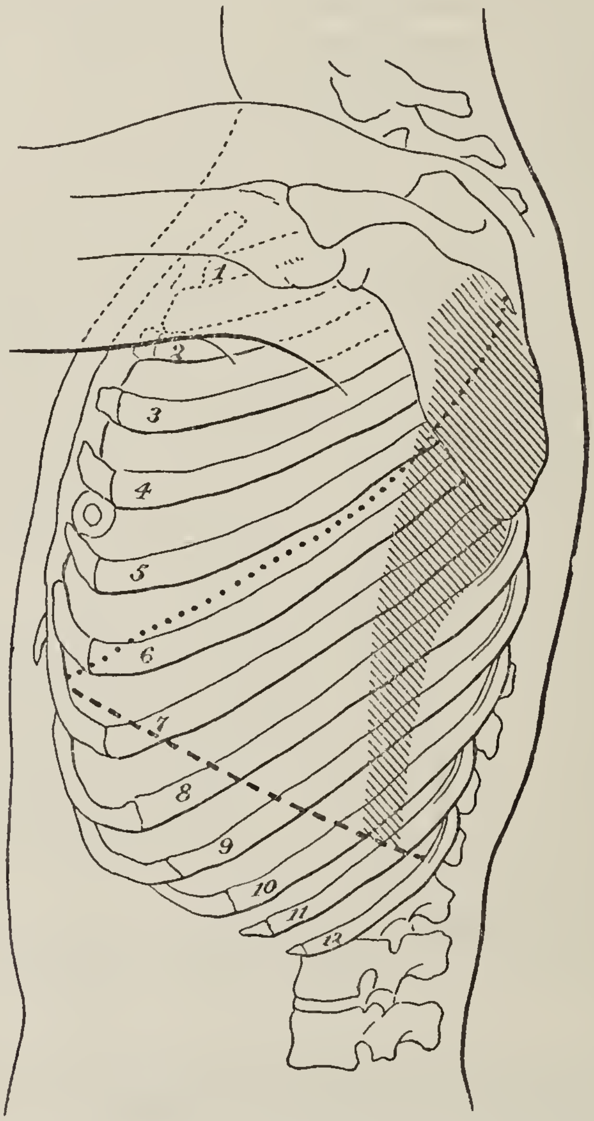

FIG. 136.—Case XV. Large, but distinctly encapsulated empyema.

November 8. Operation: Eucain 2 per cent. Patient prone. Resection of 3 cm. of 9th right rib in posterior axillary line. The sinus of empyema runs up posteriorly to the region of the third rib, where the cavity is larger. The lung is bound down tight by adhesions (Fig. 136).

November 9. Temperature normal.

November 18. Tube out.

November 23. Discharged.

September 28, 1915. Refuses to return for examination. Wife says he is in good health but has a cough.

CASE 16.—Empyema, left. Recovered.

Joseph R., nineteen years.

December 3, 1914. Admitted to Dr. Frazier's service, Episcopal Hospital. Pneumonia (left) three weeks ago; crisis two weeks ago but did not get well. Fever developed with pain in left chest and dyspnea. Cough and expectoration continued. Needle in 7th interspace shows pus. (Family physician.)

Examination on admission: Left base, absent breath sounds and fremitus; and flatness. Hoover's sign positive. White blood cells, 15,840. Temperature, 103° F.

December 4. Operation: Eucain 2 per cent. Excised 2.5 cm. of 8th left rib posteriorly. 500 c.c. of pus (diplococci).

December 8. Tube lost in wound (was sutured to skin by No. 1 chromic gut). Roentgen ray 3376*a* shows it inside chest.

December 11. Efforts yesterday failing to extract tube, Dr. Ashhurst today (gas) resected 2.5 cm. more off same rib at anterior and then at posterior end of former section, and finally found tube in spinal gutter posteriorly. *Large pus pocket* opened and drained. Tube fastened with silkworm gut and safety-pin. Temperature has been normal since operation last week.

December 14. Drainage has about stopped.

December 18. Out of bed.

December 22. Discharged.

September 28, 1916. Has removed to Wilkes-Barre, Pa.

CASE 17.—Pulsating empyema necessitatis, left. Recovered.

Garald L., twenty-eight years.

December 10, 1914. Admitted to Dr. Frazier's service, Episcopal Hospital.

Past History. Is tuberculous; has been ill off and on for seven years. In February, 1911, had left pneumonia, followed by pleural effusion (empyema), tapped, tube inserted (no rib resected). Acute illness lasted six months. Sinus ran nearly one and a half years. Recovered and resumed work about one and a half years ago (laborer).

Present Illness. Ailing for one month, very ill for one week, high fever, bad cough, pain in chest; expectoration offensive and profuse.

Examination: Extremely ill, delirious, cyanosed and dyspneic. Empyema necessitatis over second left rib and in upper axilla. Former pulsates synchronously with heart. Above middle of scapula there is Skodaic resonance; below this flat, immobile (Hoover's sign very pronounced); breath sounds and fremitus absent. Over apex (L) breath sounds loud and blowing, almost amphoric. Right lung full of crepitant rales. White blood cells, 50,000. Polynuclears 91.3 per cent. Temperature 99°. Pulse 100 to 128. Diagnosis: Pyopneumothorax. Heart displaced 4 cm. to right of sternum. Thirty-eight ounces (well over one liter) of pus removed by aspiration in left 8th interspace posteriorly.

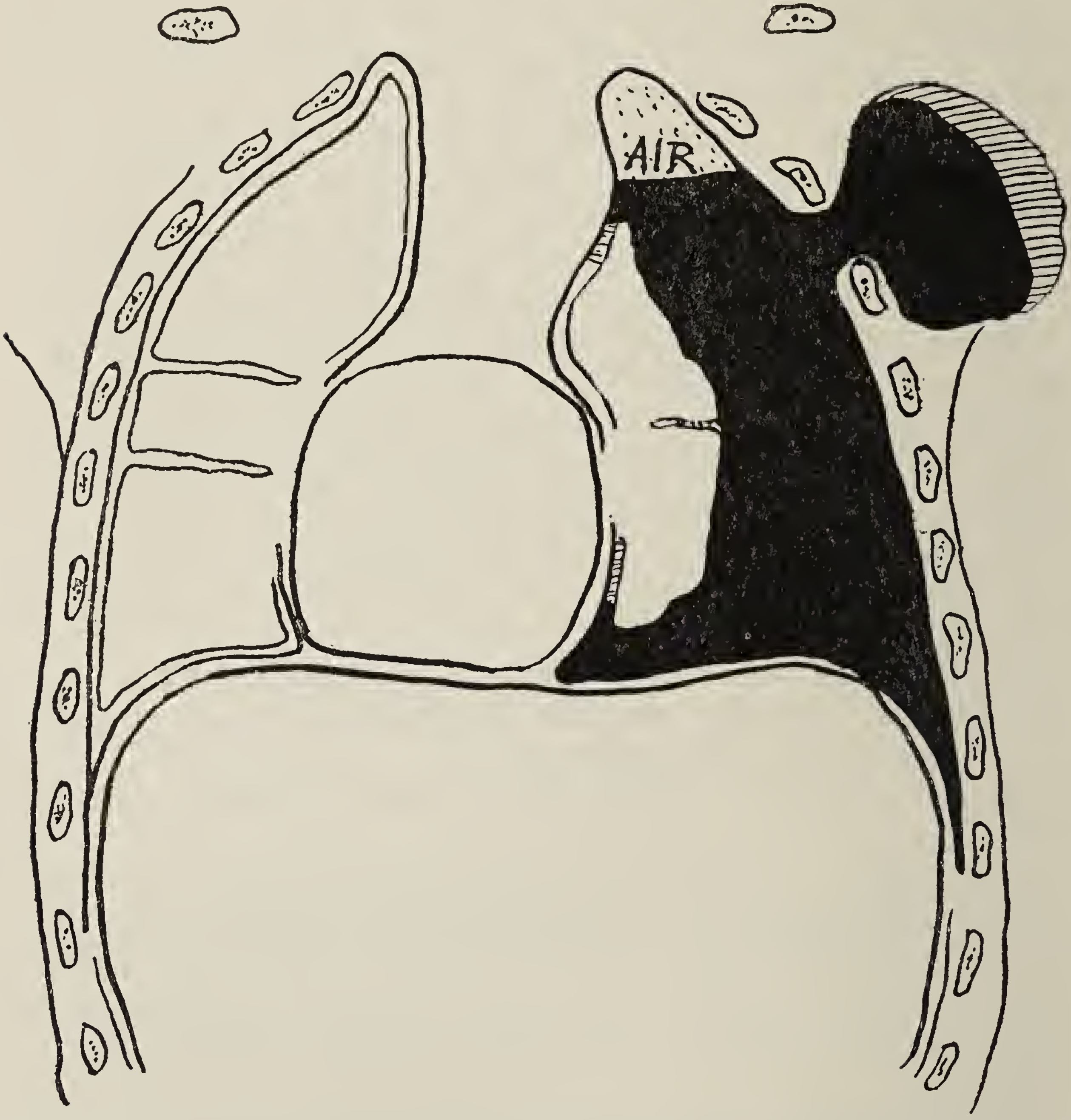

FIG. 137.—Case XVII. Pulsating empyema necessitatis (pyopneumothorax.)

Foul and fecal smelling. (Long pale bacillus with streptococcus predominating.) After aspiration bulging area disappeared below clavicle, and this area was sucked in with each respiration. Given morphin for cough.

December 11. Still in poor condition but much better than on admission.

Operation: Eucain, 2 per cent. Excised 3 cm. of 9th rib (L). Patient lying prone. Pleura very thick (5 mm.) and prune-juice looking pus evacuated after breaking up some adhesions. Exceedingly bad stink of hydrogen sulphide gas. Long tube inserted.

December 12. Better; marked discharge. Temperature, 102°.

December 14. Temperature again normal.

December 18. Tube removed.

December 29. Went home against advice, but in very good condition; out of bed several days.

September 28, 1916. Has moved to Souderton, Pa.

Case 18.—Empyema right (encysted). Recovered.

Tony V., two years.

November 15, 1915. Admitted to Dr. Frazier's service at the Episcopal Hospital.

Three weeks ago became ill and treated at home for pneumonia; seen today by Dr. R. S. Hooker at home and diagnosed as empyema. On admission temperature 102°. Pulse, 108 to 148. Respiration, 32 to 40. White blood cells, 25,000. Poorly nourished; thin profuse nasal discharge. Pharynx unusually red. Thorax: resonance good, a few rales low in right axilla, and breath sounds are harsh around angle of both scapulæ.

November 11. Aspiration (numerous punctures) of right pleural cavity; no fluid obtained. Temperature down.

November 13. Temperature up.

November 15. Aspirated again in left axilla; no fluid obtained.

November 16. White blood cells, 36,640. Lymphocytes, 40 per cent. Polynuclears, 46 per cent. Hemoglobin, 59 per cent.

November 17. Temperature down.

November 18. Temperature up.

November 19 to 23. Temperature, 100° to 103°; irregular. Seen in consultation by Dr. W. E. Robertson, who diagnosed fluid under the 6th rib right. Roentgen ray confirms this.

November 23. Operation by Dr. Ashhurst (ether). Time ten minutes. Patient prone. Resection of 3 cm. of 7th rib near angle of scapula, pleura thick. Opened this, no pus. Finger introduced found lung lightly adherent to parietal pleura and diaphragm all over. No dense adhesions. No interlobar collection between middle and lower lobes found. Only blood. (Culture: sterile.) Drain: rubber tube.

November 26. Wound doing well.

November 29. Temperature has fallen gradually to normal.

December 1. Sudden profuse discharge of pus through wound.

(Culture: streptococci and staphylococci.) Evidently an encysted empyema had broken into wound. Temperature, 101° F.

December 3. Considerable drainage.

December 8. Discharge scanty.

December 12. Chest still draining but scanty.

February 5, 1916. Went home. Sinus healed dry.

September 6, 1916. No deformity, lung normal, rib reformed.

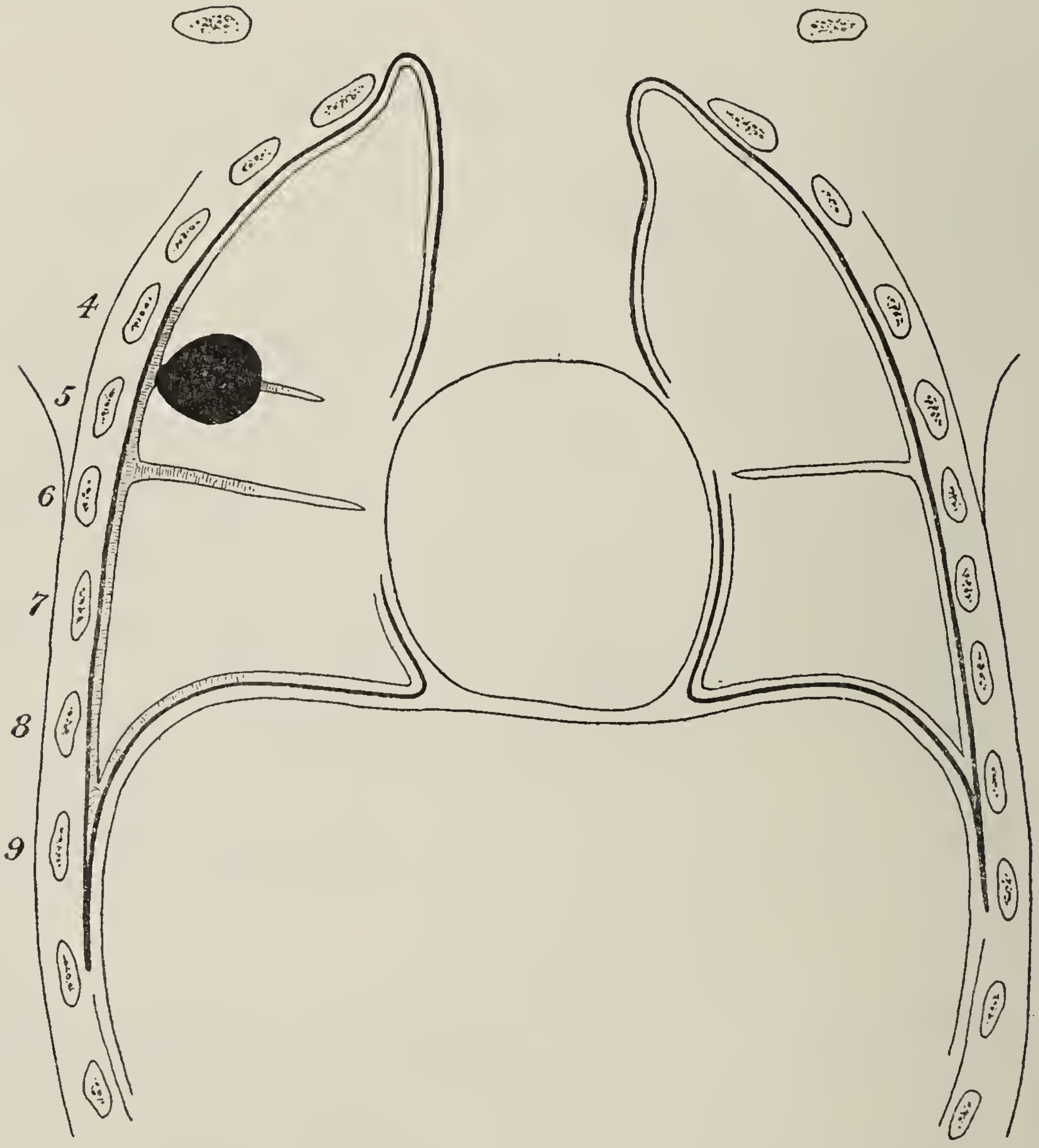

Fig. 138.—Case XVIII. Empyema encysted between upper and middle lobes of right lung; not found at operation. Later ruptured into drainage tract.

Case 19.—Empyema right. Recovered.

Joseph F., nine years.

November 17, 1915. Admitted to Dr. Stevens's service (P. E. H.).

About four days ago became sick, attributed to eating some cabbage two days before. Pain in abdomen and then vomited;

pain was continuous. This morning pain seemed localized to upper right abdomen and patient then began to show rapid and shallow respirations and to dart his tongue in and out. Has eaten very little since illness began, but has not been constipated.

On admission heart not enlarged; no murmus. Lungs anteriorly resonant throughout; breath sounds normal posteriorly; dulness and absent breath sounds over right lower lobe up to angle of scapula. Diminished resonance over left lower lobe (Grocco's sign). Few moist rales above area of dulness on right. Liver not enlarged; great tenderness and much rigidity in right upper quadrant; no mared tenderness in right iliac fossa.

November 18. White blood cells, 25,000.

November. 27. Diagnosis of empyema made and transferred to surgical service. Temperature has been 102° to 103° ever since admission, growing more irregular (99° to 104°) since November 23.

November 28. 500 c.c. of colon smelling pus removed by aspiration. Dyspnea much relieved. Culture of pleural fluid; mixed growth.

November 30. Operation by Dr. Ashhurst (gas). Patient supine, right chest projecting over edge of table. Resectnd 4 cm. of rib below angle of scapula, evacuating nearly 500 c.c. of colon pus; undoubtedly *not* subphrenic but in pleural cavity. No perforation of diaphragm felt. Drainage tube. Temperature fell at once.

December 3. Draining freely.

December 12. Drainage much less.

December 22. Out of bed, temperatureu still down.

January 3, 1916. Went home. Small tube still in sinus and some discharge.

August 29, 1916. Examination in dispensary. Very slight scoliosis; sinus healed by February 1, 1916. Lung sounds normal.

CASE 20.—Empyema, recurrent (right). Recovered.

Francis P., eleven years.

October 13, 1915. Admitted to Dr. Deaver's service (P. E. H.). Two and a half weeks ago developed pneumonia, for past five days excessive cough and profuse expectoration. On admission temperature, 101°. Pulse, 112. Respiration, 36. Respiration shallow. Right chest more prominent and motionless on breathing. Flatness over whole right chest, feeble breath sounds, tubular on deep breathing. Left chest hyperresonant, few rales. Heart displaced to left, apex in 5th interspace, anterior axillary line. By aspiration, 5 c.c. foul greenish pus; staphylococci, diplococci and streptococci.

October 14. Operation (Dr. Deaver). Ether. Resected 2.5 cm. of 7th rib left midaxillary line. 200 c.c. of pus. Rubber tube.

October 24. Still profuse discharge. Temperature, 99° to 103°.

October 29. White blood cells, 14,640. Lymphocytes, 44 per cent.

November 1. Transferred to Dr. Frazier's service. Dressings still require changing twice daily; tube still in. Temperature, 99° to 101°.

November 8. Dressed once daily.

November 17. Drainage less.

November 26. Temperature up.

November 29. Temperature still up.

December 3. Second operation (Dr. Ashhurst). Ether. Old sinus excised by incision in long axis of patient's body. Excised 3 cm. of rib next above and 3 cm. more off anterior end of that formerly excised (almost closed). Burrowed between adherent lung and thoracic wall posteriorly, gush of fetid thin pus. (Culture: streptococcus.) About 150 c.c. Rubber tube drain.

December 5. Foul discharge (Bacillus pyocyaneus).

December 12. Improved.

December 13. Seen by Dr. Stevens, who diagnosed pus in extreme lower part of right pleural cavity. Circulation not very good.

January 14, 1916. Sinus is being exposed to sun daily.

January 24. Temperature much lower, drainage less.

February 1. Discharge very slight. Breath sounds good.

February 3. Discharged from hospital.

September 13, 1916. Robust and rosy. No deformity. Sinus healed solidly since March. Rib reformed. Thickened pleura, but lung normal. (Impaired resonance and fremitus and distant breath sounds.)

Case 21.—Empyema left. Encapsulated, with separate pleural effusion. Recovered.

Joseph B., thirty-four years.

December 8, 1915. Admitted to Dr. Piersol's service; Episcopal Hospital.

Present Illness. Three weeks ago pain in left chest and cough. On admission temperature 100°. Pulse, 92. Respiration, 20 to 24. White blood cells, 22,000. Polynuclears, 89 per cent. Pain in lower left chest; slight cough. Not toxic. Not dyspneic. Bulging of left anterolateral chest wall; dull red, extremely tender and resistant, from anterior axillary line into axilla. Respiratory excursions limited over left lower thorax; vocal fremitus absent, anteriorly below level of nipple and in left axilla below this level; and posteriorly below angle of scapula. Skodaic resonance over upper left lung. Traube's semilunar space seems obliterated. Flatness over left chest below nipple, also absent vocal resonance, and breath sounds inaudible.

Right lung: rough puerile breathing throughout and dry rales.

Left chest tapped posteriorly, turbid straw-colored fluid, not true pus. (Laboratory report: Gelatinous, 320 cells per c.mm.; 80 per cent. polynuclears; 20 per cent. lymphocytes.) Blood Wassermann negative.

December 9. Seen in consultation by Dr. Ashhurst. Evidently an extrathoracic abscess over left anterolateral chest and inflammatory exudate in pleura. Transferred to surgical service.

Operation by Dr. Ashhurst (novocain $\frac{1}{4}$ per cent.).

(*a*) Resected two inches of left 11th rib posteriorly; pleura opened; profuse discharge of slightly turbid fluid. No odor. Diaphragm comes up against wound in expiration and fluid only discharges when diaphragm is pushed away. Finger introduced finds no adhesions where diaphragm comes against ribs. Lung not felt. About 300 to 350 c.c. of turbid fluid evacuated and more drains while dressing wound. Tube inserted up four inches and stream of turbid serum escaped through it. Tube sutured to skin and ends of skin wound closed. (Culture of serum: negative.)

(*b*) Patient on back. Novocain. Incision over subcutaneous (left) phlegmon; thick yellow inodorous pus (culture: Staphylococcus). This pus had spread over area 8 cm. in diameter superficial to ribs and discharged between 7th and 8th ribs through orifice admitting little finger, into walled-off cavity about 8 cm. in diameter; encapsulated empyema necessitatis. Iodoform gauze drain. (Query: Is free pleural fluid tuberculous? Presumably it is analogous to that in pleura in subphrenic abscess and to that in knee in osteomyelitis of tibia.)

December 10. Temperature 80° to 103° F.
December 15. Temperature steady 98° to $99\frac{2}{5}°$.
December 25. Tube out from posterior wound.
December 29. Out of bed.
January 7, 1916. No drainage. Went home at own request.

September 20, 1916. Examination: Returned to work two months after leaving hospital. No disability since. Both wounds solidly healed, lung normal on examination.

CASE 22.—Empyema, right; necrosis of mandible (from noma). Died two days (noma).

Edward S., seven years.

December 11, 1915. Admitted to Dr. Frazier's service (P. E. H.).

Pneumonia at home for three weeks. December 7th refused to eat because of sore mouth, and for four days before admission was pulling out loose molar teeth with his fingers; had very foul breath. Entire alveolus of lower jaw (right and left), except under the three front teeth, is necrotic, and the noma extends on to cheeks. Right lung: dull except over extreme apex, breathing tubular and fairly loud where dull, and fremitus increased. Left lung: numerous rales and tubular breathing at base.

1 P.M. Red blood cells, 2,800,000. Hemoglobin, 35 per cent. White blood cells, 15,800. Temperature, $101\frac{2}{5}$°. Condition very poor. Aspiration right chest, 10 c.c.; thick yellowish pus.

2 P.M. Child desperately ill from sepsis.

5 P.M. Operation by Dr. Ashhurst (gas). Resection of 3 cm. of 8th right rib in posterior axillary line; patient on back. Pleura not injured. On opening it (rather thick) a squirt of thick creamy yellow pus, no odor. (Culture: mixed growth.) About 300 c.c. evacuated. Cavity extends three ribs lower to costophrenic space and up as far as finger can reach toward apex. Respirations very bad. (Inhalations of ammonia.) Rubber tube drain. Then fuming nitric acid to mouth, and alveolus of mandible excised.

December 13, 1915. Died 1 A.M.

CASE 23.—Empyema left. Recovered.

James C., three years.

January 4, 1916. Admitted to the Episcopal Hospital, Dr. Ashhurst's service. Three weeks ago became ill with cough, high temperature and pain in left side. On admission: Respiration rapid, expansion absent on left. Fremitus is diminished and breath sounds almost absent; entire left chest is dull on percussion. Apex-beat in midline. White blood cells, 16,640. Polynuclears, 71 per cent. 500 c.c. pus drawn by aspirator. No dyspnea before or after. Report from laboratory January 5: Pneumococcus.

January 7, 1916. Operation by Dr. Ashhurst (ether). Patient prone, resected 10th left rib in posterior axillary line. 250 c.c. of creamy pus evacuated; no masses of lymph. Loosened lung from diaphragm and from spinal gutter, whereupon it expanded 50 per cent.

January 17. Drainage much less.

January 22. Temperature up, slight cough.

January 26. Developed measles and sent to Philadelphia General Hospital.

February 3. Returned from Philadelphia Hospital. Temperature, 100° to 101°.

February 6. Temperature, 104°. Draining freely.

February 15. Temperature falling.

February 26. Tube removed.

March 1. Tube replaced.

March 6. Tube removed. No drainage.

March 10. Discharged. Sinus healed.

September 6, 1916. No scoliosis, ribs reformed, perfect result.

Case 24.—Empyema, encysted, left. Recovered.

Philip G., fourteen years.

December 27, 1915. Admitted to Dr. Piersol's service at the Episcopal Hospital.

Two weeks ago developed chill, headache, vomited several times, and had generalized slight pain. After few days felt better and went out. Then right ear began to ache; and two days ago developed cough, and became constipated. Has now coryza, cough, slight dyspnea and is constipated. No appetite since illness began.

On admission: fairly well built, slender, not emaciated, no adenopathy. Heart is not enlarged nor displaced, apex-beat in usual location, no murmurs, sounds of fair quality. Lungs are negative, good breath sounds, good resonance throughout. Abdomen negative. White blood cells, 11,040.

December 30. Marked diminution in breath sounds over left base posteriorly, few sonorous rales in lower left axilla. Sputum negative for tubercle bacilli.

January 2, 1916. Widal negative.

January 4. Marked bronchial breathing over right apex posteriorly.

January 6. Blood culture: short chain streptococcus. Malaria negative. White blood cells, 19,960.

January 12. Dulness on percussion and great tenderness at left base posteriorly. Nothing obtained on exploratory puncture.

January 13. Examined by Dr. Ashhurst in consultation: Has been in medical ward over two weeks with septic temperature (100° to 104°) and chills. Patient is extremely emaciated and anemic. Left lung dull at base, all signs of empyema but needle found no pus. Very tender in left costovertebral angle. Excursion of costal margin from midline (Hoover's test) not increased, no more tender in left than in right hypochondrium.

Roentgen ray (only properly interpreted after operation) shows dense shadow above left diaphragm, upper level horizontal (not domed) and higher than diaphragm on right. A diagnosis was made of encysted empyema and exploratory operation advised.

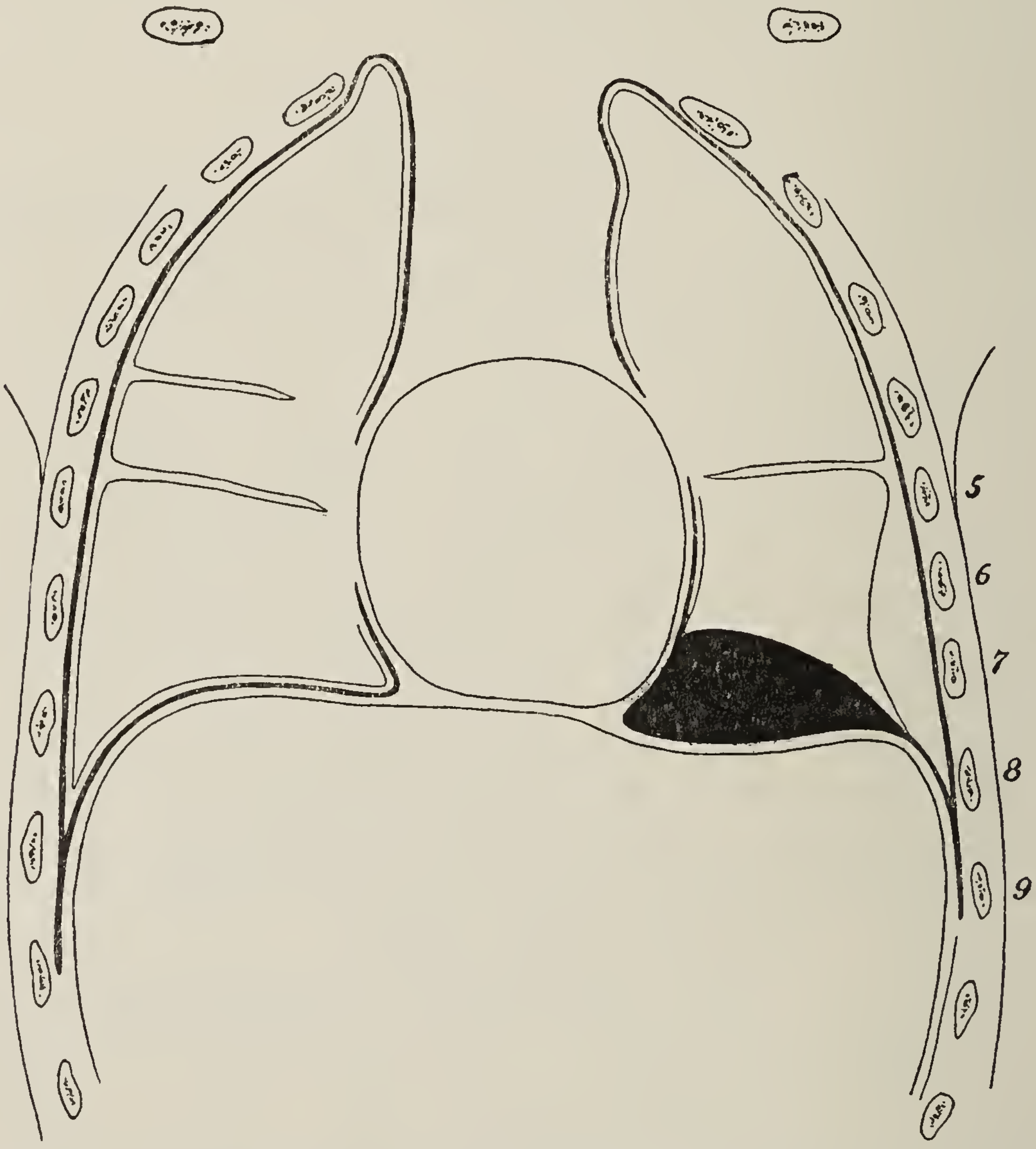

Fig. 139.—Case XXIV. Empyema encysted between left lung and diaphragm. Cases XXVII and XXXVI were quite similar, but on the right side.

January 14. Operation by Dr. Ashhurst. Stovain locally for resection of rib; gas for intrathoracic explorations. Patient prone. Excised 5 cm. of 10th rib near angle. Then incised pleura. Lung did not collapse. Diaphragm exposed, soft; spleen easily palpated through diaphragm, no sign of subphrenic abscess, and on retracting upper ribs (now general anesthetic) lung was seen to be plastered on to diaphragm. Extended incision up parallel to spine, excised 6 cm. more of rib at vertebral end. Dissected carefully with finger, raising lung from diaphragm; a flood of pus (500 c.c.) (culture: streptococci) from between lung and diaphragm. Lung now collapsed. Drain, rubber tube and iodoform gauze between lung and diaphragm. Ends of incision (12.5 cm.) closed with silkworm gut.

January 15. Given enteroclysis and liquids by mouth. Looks better, quiet, comfortable, but very weak and thin.

January 17. Temperature, 98° to 100° ever since operation. Eating better. Looks better.

January 22. Tube shortened.

January 25. Tube removed. Rapidly improving.

January 31. Up in chair.

February 14. Able to walk.

February 26. Sent home.

August 9, 1916. In dispensary in good general health, sinus in wound just admits probe; has been dressed three times weekly by family physician since leaving hospital; tube was retained until last week, since when gauze has been employed. It was recommended that the packing be discontinued.

February 29, 1920. Sinus closed permanently a week after the gauze packing was discontinued. The only symptom he complains of now is a pricking in the scar at times on exertion. He works as meat-chopper, lifting heavy portions of the carcasses without disability. There is no scoliosis and no deformity except the very much depressed scar. The lungs are normal, but the ribs below the scar do not move in forced respiration as do those on the other side.

Case 25.—Empyema, encysted, right. Recovered.

William B., thirty years.

December 22, 1915. Admitted to the Episcopal Hospital, Dr. Piersol's service.

Six weeks ago developed pain in back and slight dyspnea; began to cough, with slight mucopurulent expectoration. Yesterday pain in right side at costal margin, cough, some dyspnea, rapidly becoming worse. On admission: temperature, 103°. Pulse, 104 to 108. Respiration, 32. White blood cells, 10,000. Polynuclears, 91 per cent. Well developed, dyspneic, alæ of nose moving in each respiration. Lungs: Impairment of resonance over right side, most marked in middle and lower lobes; tactile and vocal fremitus decreased and bronchial breathing with moist rales over middle and lower lobes. Few sonorous and crepitant rales over right upper lobe. Left lung negative. Heart normal. Liver enlarged 5 cm. below costal margin.

December 26. Temperature fell by lysis to 100°.

December 30. General condition improved. Temperature, 100° to 103°. Marked bronchial breathing over middle of right lung posteriorly.

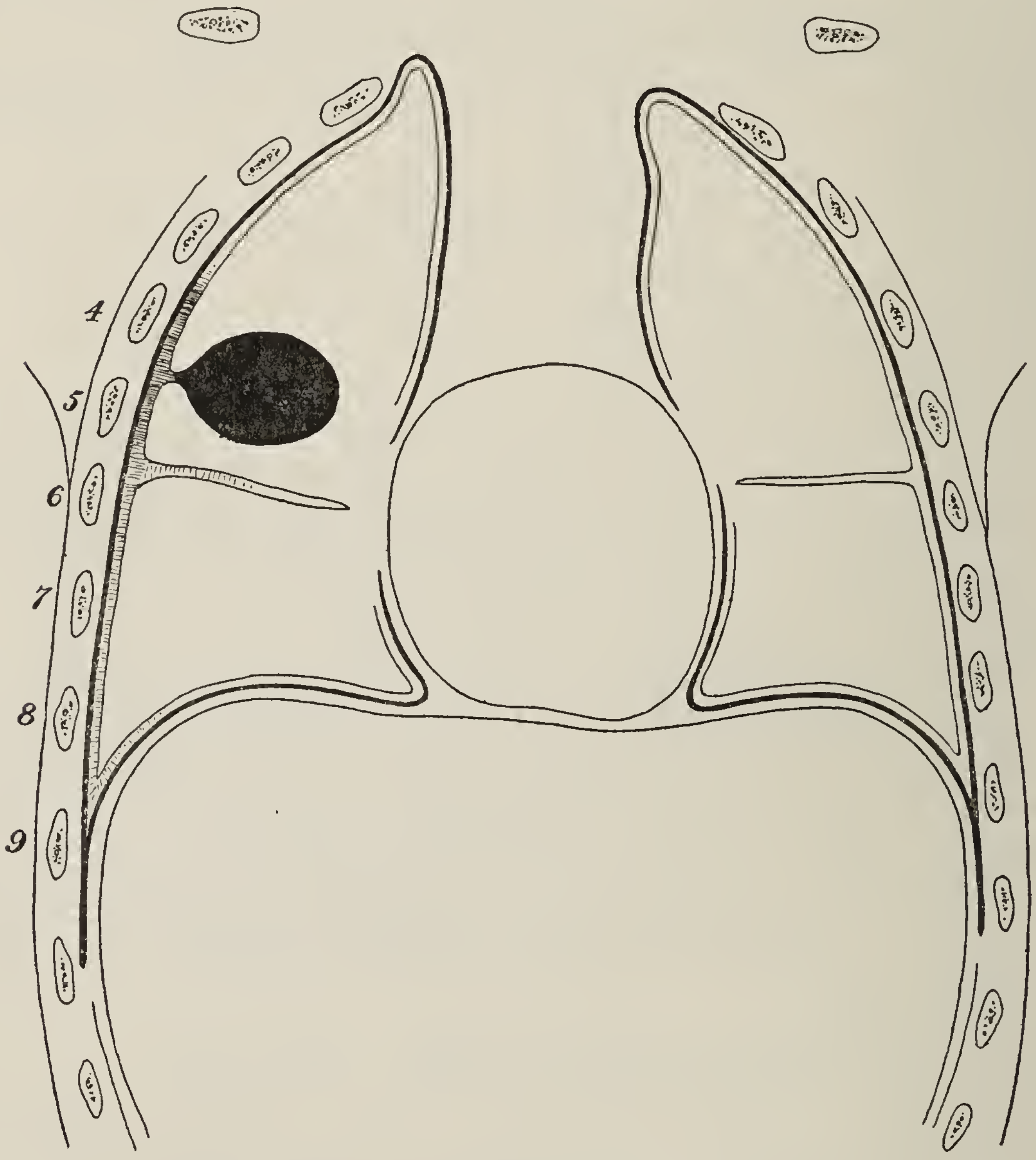

FIG. 140.—Case XXV. Empyema encapsulated between upper and middle lobes of right lung. Evacuated by thoracotomy. Compare Fig. 13 (Case XVIII).

January 3 to 14, 1916. Temperature, 99° to 101° to 102°, irregular.

January 13. Seen by Dr. Ashhurst in consultation. In medical ward (Piersol) some weeks with sepsis following pneumonia ("unresolved"). Examination: Right chest dull (flat) from angle of scapula down, fremitus normal, breath sounds distant. A diagnosis of empyema was made from history and condition of "unresolved pneumonia." Medical attendants could not locate pus. Needle in 8th interspace drew only blood.

January 14. Transferred to surgical ward. Dulness over middle and lower lobes right. Right base, however, transmits vocal fremitus, although voice and breath sounds are diminished. Roentgen ray shows haziness of upper and middle lobes. Operation by Dr. Ashhurst (novocain $\frac{1}{4}$ per cent.). Patient prone. Incision 10 cm. over 9th rib, excised 8 cm. Diaphragm and lightly adherent lung present on opening pleura. Separated lung from diaphragm by finger as far as could reach; no pus. Surface of diaphragm rather sensitive; lung insensitive. Then burrowed between middle and lower lobes; no pus; entirely insensitive. Then burrowed up along parietal pleura and found very dense adhesions up along 5th to 6th ribs, finally broke through, when just about to abandon operation and got a flood (perhaps 250 c.c.) of blood-stained pus. (Culture: pneumococcus). Evidently had been interlobar (upper and middle lobes) and had worked out to parietal pleura. Parietal pleura rather sensitive. Slight coughing on evacuation of pus and partial collapse of lung. Drain: rubber tube up along parietal pleura for four inches.

January 15. Looks convalescent.

January 16. Drains freely.

January 20. Tube removed.

January 23. Temperature normal. Out of bed. Sputum negative for tubercle bacilli.

January 31. Convalescent.

February 3. No discharge. Small granulating wound. Sent home.

August 9, 1916. Cannot be traced.

CASE 26.—Interlobar empyema; left (against pericardium). Died.

Katherine D., thirty-eight years.

August 3, 1916. Admitted to Dr. Ashhurst's service (P. E. H.).

Previous History. Four and a half years ago had attack of pneumonia and pleurisy (left sided) and was in another hospital five months; had septic temperature and chills. Was aspirated but no pus could be obtained.

Present Illness. Has had no trouble since until six weeks ago, when she had pain in left side for two days, severe pain for three-quarters of an hour, followed by chill. July 28 had temperature 103°, pulse 120, and sent for Dr. Shannon. Has had chills. August 1 went to bed complaining of weakness and fever and pain on left side. Last night (August 2 and 3) had pain in right shoulder, relieved by hot-water compresses. Has had slight cough but not much expectoration. Coughed more before taking to her bed.

On admission: well nourished, no evidence of acute suffering. Breath foul. Temperature 101° to 105°. Pulse 120 to 150. Respiration 32 to 40. White blood cells 21,000. Chest: Posteriorly inspection and palpation negative except for slight restriction of expansion and decreased fremitus at base of left lung. Percussion note is impaired from 7th interspace in mid-axillary line to vertebral column and extending to base of left lung. Auscultation shows decreased breath sounds over this area. Right lung negative; breath sounds normal. Heart: Rapid; no murmurs; outline normal. Abdomen negative. Punct-

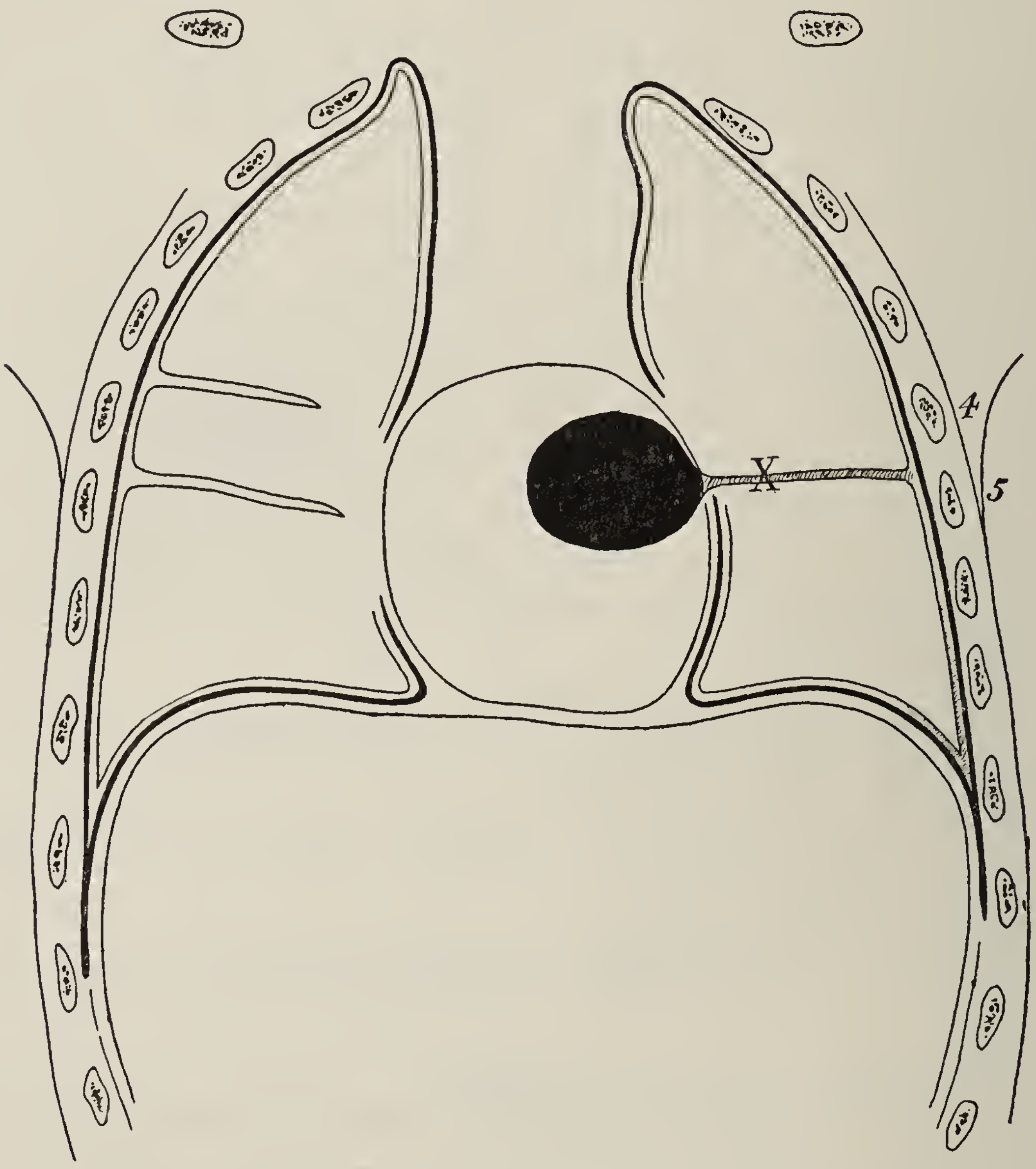

FIG. 141.—Case XXVI. Empyema encapsulated between fissure of left lung and pericardium. *X* indicates the point reached at operation.

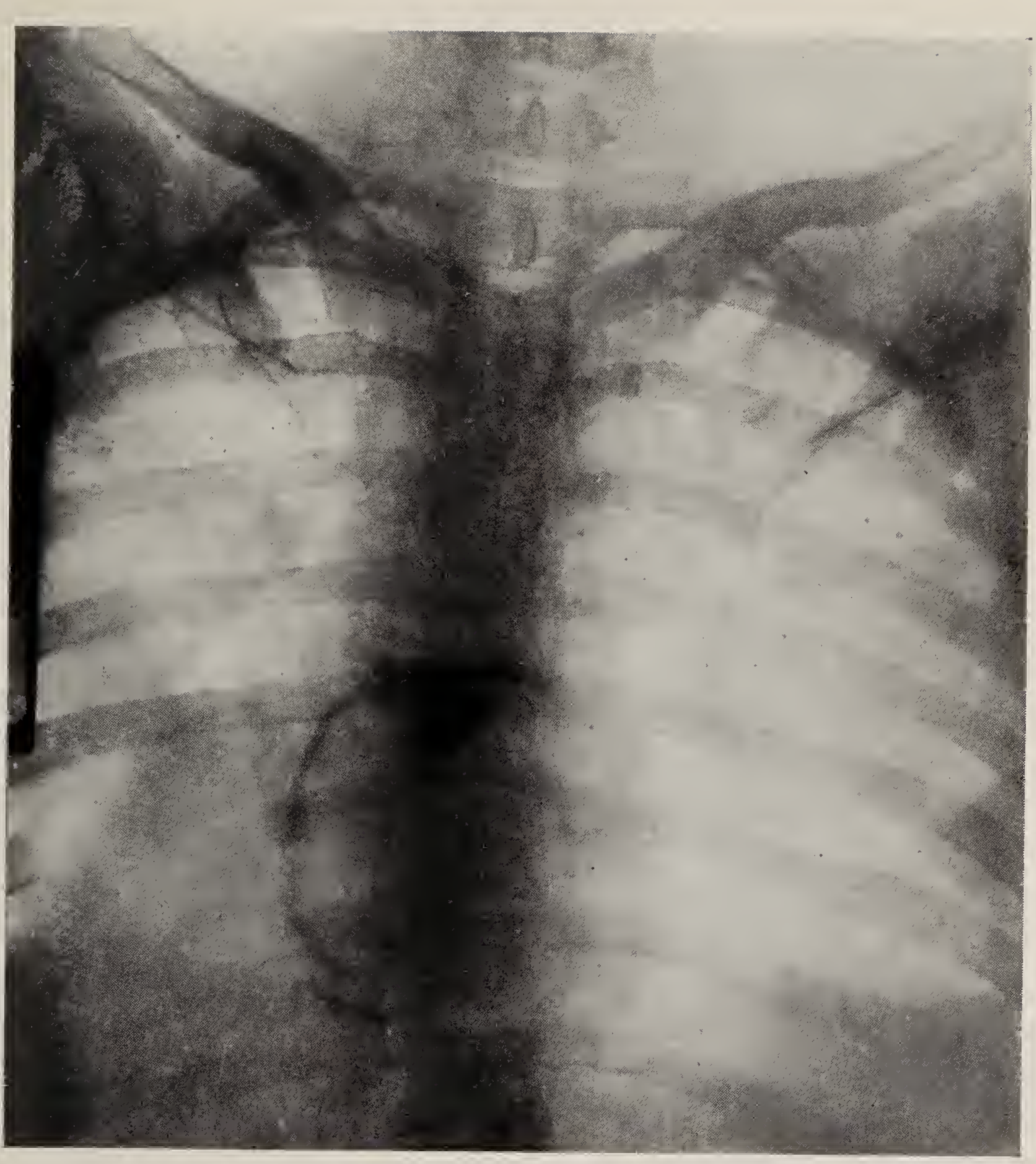

FIG. 142.—Case XXVI. Encysted empyema three days after operation at which no pus was found. Note distinct outlines of abscess cavity overlying shadows of sixth, seventh and eighth thoracic vertebræ, mostly to left of midline. (See Figs. 141 and 143.)

ure in 7th interspace, anterior axillary line (Record syringe) gave no fluid. After admission had chill.

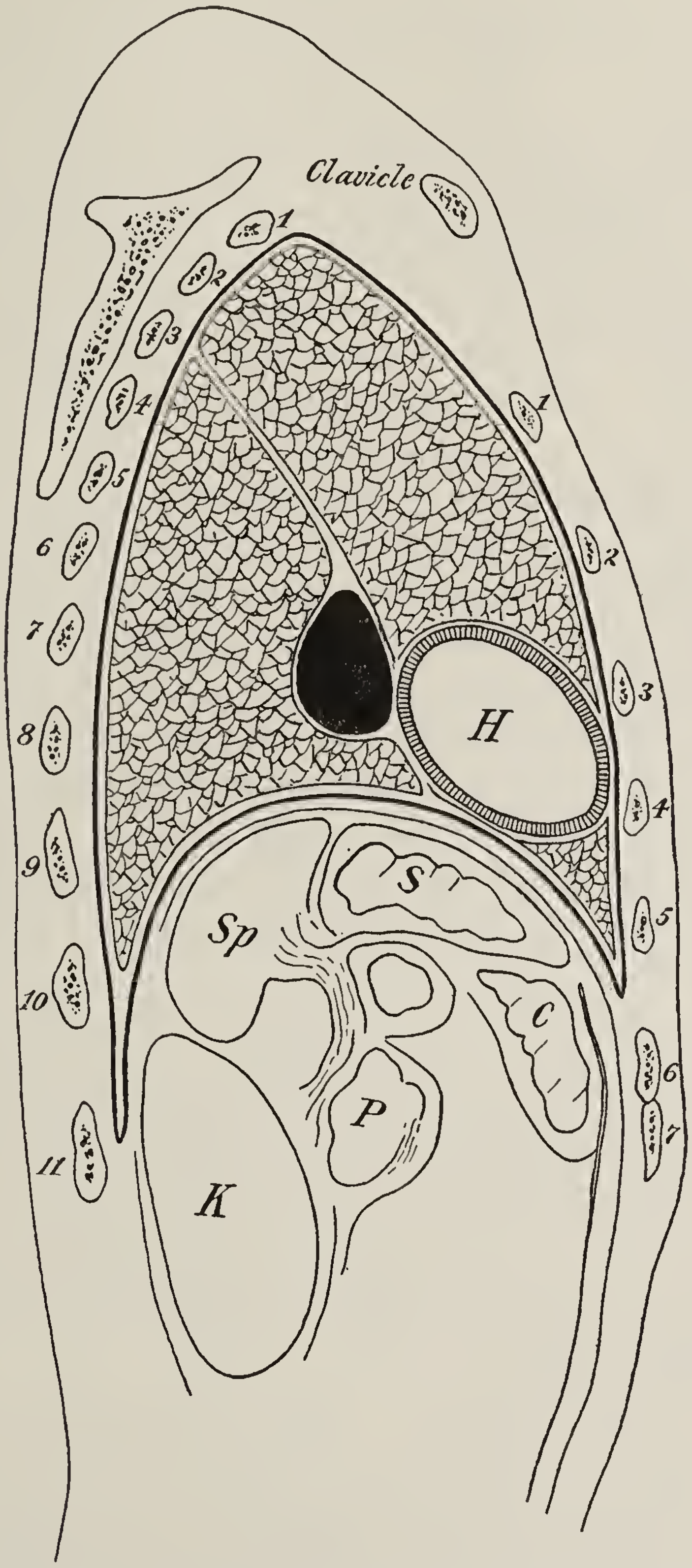

FIG. 143.—Case XXVI. Diagram of sagittal section through middle of left clavicle, indicating site at which pus was found at autopsy.

August 4. Another chill today. Temperature 101° to 104°. Roentgen-ray apparatus out of order and no roentgenological examination could be made.

August 5, 1916. Operation by Dr. Ashhurst (novocain ¼ per cent.). Patient prone. Incision 10 cm. over left 8th rib, excised 8 cm. Opened pleura, lung lightly adherent throughout but freely movable. Lung densely adherent at periphery to diaphragm. Packed off above. Dissected lung from diaphragm. No pus. Felt inner border of spleen through diaphragm: no subphrenic abscess. Packed over diaphragm, removed upper packing and separated lung from parietal pleura. Then extended skin incision, excised 3 cm. more of 8th rib and 10 cm. of 7th; dissected with two forceps in interlobar space for 5 or 6 cm. but found only dark blood. (Culture: Sterile.) Iodoform gauze stuffed in this hole and wound closed in layers. (Hoped pus would break into wound as in Case XVIII.)

August 7. Examination by Dr. Hooker. On left side down to angle of scapula there are normal breath sounds, resonance and fremitus. Unable to examine below on account of dressing. Heart, second aortic accentuated, no murmurs. Blood culture negative. One chill today. Temperature 98.4° to 104°.

August 9. Examination by Dr. Hooker: Heart, second aortic accentuated, roughening at mitral valve, presystolic. Entire right lung shows increased vocal resonanace, many crepitant rales, impaired note on percussion, typical pneumonia (hypostatic in origin). Left lung same as last examination. Drainage removed and wound clean.

August 10. Expression is anxious, apathetic, listless, but rather restless. Heart sounds distant. Some evidence of pus on dressings, not present yesterday. Neck held very rigid, moves extremities when attempts are made to flex neck. White blood cells, 19,000. Polynuclears, 79 per cent. Hemoglobin, 55 per cent. Temperature does not fall below 101° any more. Roentgen ray shows shadow of abscess to left of midline of thorax, under 6th and 7th ribs (Fig. 142).

August 13. Died of sepsis. Had been moribund for last three days.

Wound explored after death by Dr. H. S. Spruance (ward surgeon) after removal of two more ribs. He found gauze pack had been correctly placed in interlobar space, and by separating lobes further (about 2.5 cm.) through dense adhesions he came on abscess (100 c.c.) in interlobar space and against pericardium. Pericardium when opened was found to contain many recent fibrous adhesions but no pus. (Figs. 142, 143.)

Case 27.—Empyema left, encysted above diaphragm. Recovered.

James McG., thirty years.

October 30, 1916. Admitted to the Episcopal Hospital, Dr. Robertson's service.

Present illness began October 19 with coryza. October 28 had chill and began expectorating; continuous pain in left chest. On admittance signs of pneumonia at base of left lung.

November 5. Temperature falling by lysis.

November 11. Temperature has not reached normal. Puncture of chest negative.

November 12. Puncture again negative.

November 16. Third puncture reaches pus in 8th interspace, 40 c.c. of pus withdrawn. Smear and culture of fluid is negative.

November 17. Transferred to surgical ward (Dr. Ashhurst). Temperature 99° to 103° for the last week.

Operation by Dr. Ashhurst (eucain 1 per cent.). Excised 5 cm. of 10th rib; lung does not collapse, lightly adherent to costal pleura and densely to diaphragm. Separated costal adhesions, no pus within reach of opening. Separated lung from diaphragm as far as possible, no pus, but adhesions at limit of fingers were more dense. Excised 5 cm. of 9th rib, and after gauze pack above, dug lung away from diaphragm to dome, when about 60 c.c. of pus discharged. Drain: Rubber tube wrapped in iodoform gauze.

November 18. Temperature still up.

November 22. Still draining freely. Temperature, 100° to 103°.

December 1. Tube removed. Out of bed.

December 8. For continuing temperature and history of syphilis is given neosalvarsan.

December 12. Temperature normal.

December 18. No drainage; incision closing. Sent home.

January 16, 1917. Seen by Dr. Spruance, ward surgeon. Healed firmly for about one month. In perfect health.

February 29, 1920. Examination: Is police officer. In perfect health. Lungs normal. On palpation ribs seem to have reformed.

Case 28.—Empyema left, encysted, upper lobe. Recovered.

William B., twenty years.

October 18, 1916. Admitted to the Episcopal Hospital, Dr. Robertson's service.

Blind since illness with pneumonia at five years of age. Onset of present illness, October 16, with chilliness, during evening; awoke during night with pain in left chest. On admission: pneumonia over upper left lobe.

October 23. Temperature falling by lysis.

November 4. Temperature has not reached normal. Signs of fluid. Puncture below angle of scapula negative.

November 16. Signs of fluid still present, now confined to upper lobe, left. Puncture between angle of scapula and spine gives 30 c.c. pus. Sent to laboratory. Previous punctures all over left chest have been negative.

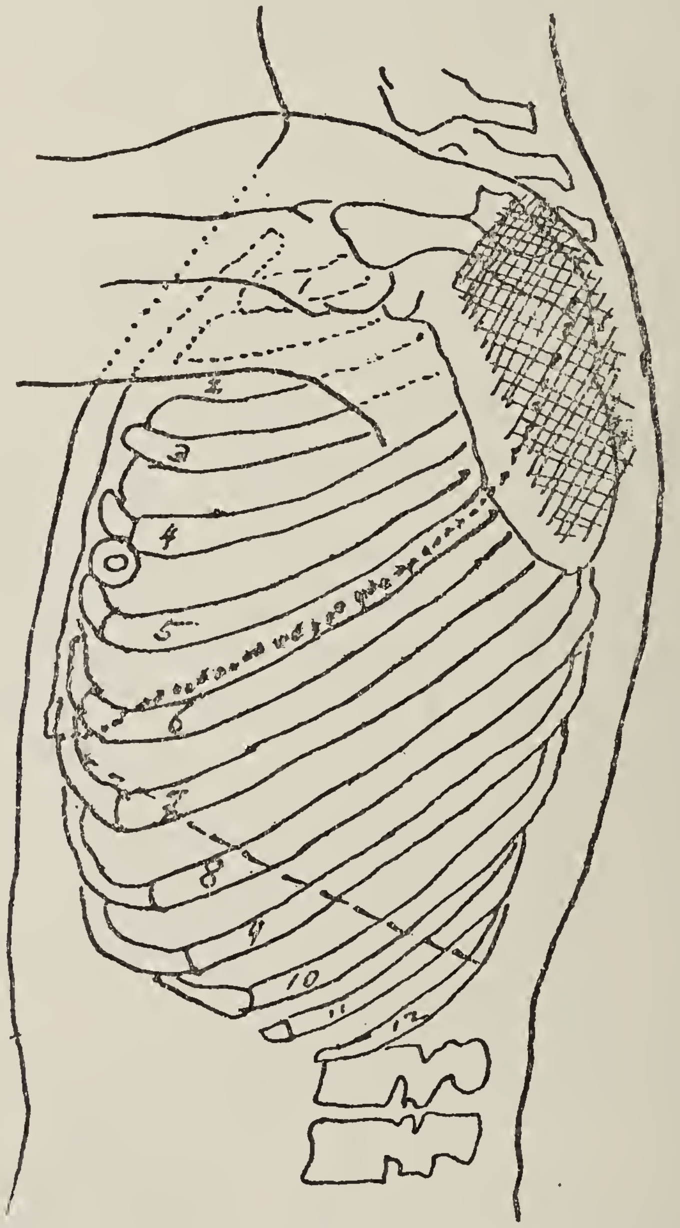

FIG. 144.—Case XXVIII. Empyema encapsulated over posterior end of left interlobar fissure.

November 17. Seen by Dr. Ashhurst in consultation. Has been in ward over four weeks, with continued temperature following typical pneumonia (left upper lobe), which declined by lysis over three weeks ago. *Repeated* punctures negative until yesterday pus was found (30 c.c. easily drawn) in fourth interspace at spine of scapula. Transferred to Surgical Service (Dr. Ashhurst).

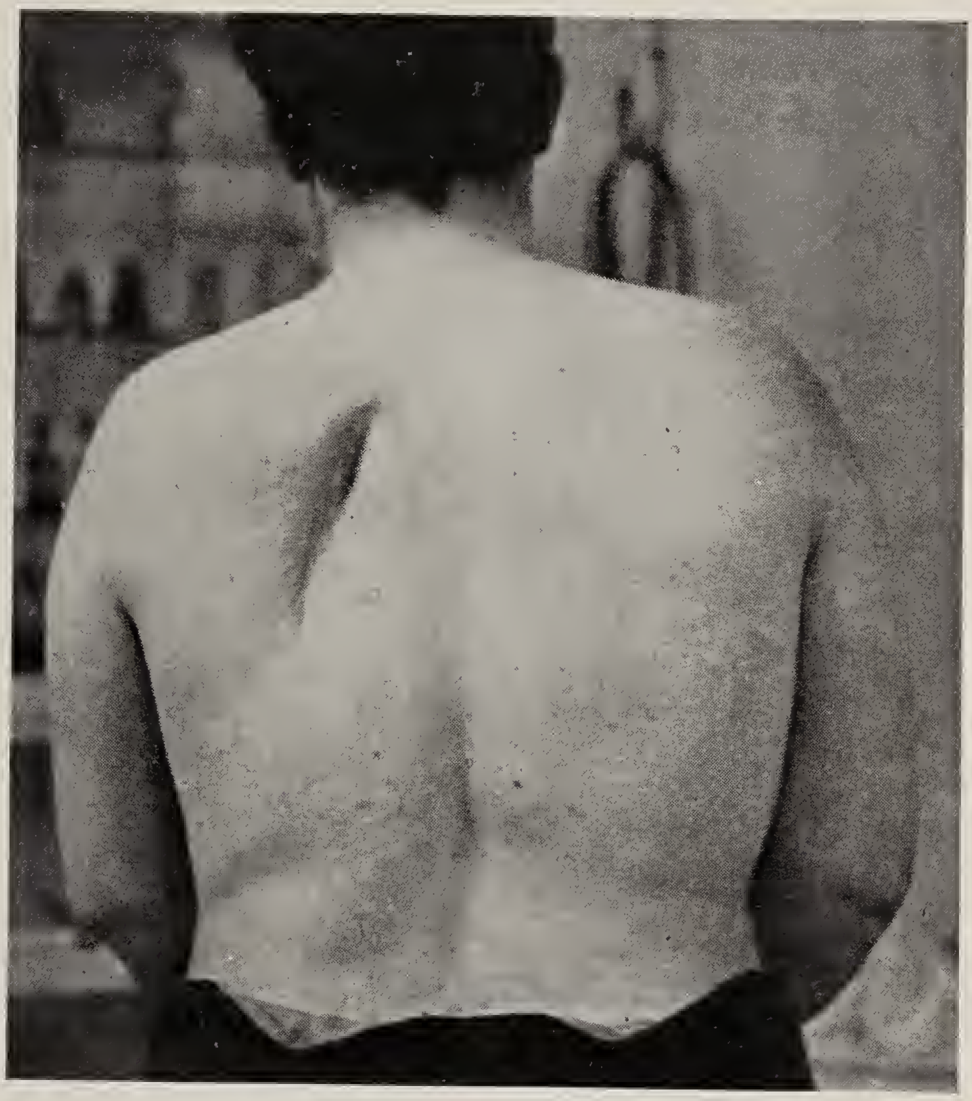

FIG. 145.—Case XXVIII. Resection of fourth and fifth ribs. Three years and a half after operation.

Operation by Dr. Ashhurst (eucain, 1 per cent.). (Patient blind since five years and neurotic.) Excised 4 cm. of fourth and fifth left ribs between scapula and spine, flood of curdy pus and great handfuls of coagulated lymph. Cavity extends to 1st rib and down only to 6th or 7th in midaxillary line, evidently encapsulated over interlobar fissure. Drain: large rubber tube and iodoform gauze.

November 18. Temperature falling, profuse drainage.

December 27. Up and about ward part of day.

December 30. Fifty million autogenous vaccines, given with idea of hastening closure of cavity.

January 8, 1917. One hundred million vaccines given.

January 16. Still in ward, not much discharge. Temperature normal for one week, until today, sudden rise.

January 26. Temperature about normal. Scarcely any discharge. Up all day.

January 27. Sent home. Scarcely any discharge.

February 21, 1920. Three years after leaving hospital, examination shows him fat and healthy. The sinus did not heal entirely for nearly ten weeks after his discharge (over four months after the operation). Examination shows the lungs normal. He has full use of his left shoulder, in spite of section of trapezius and rhomboid muscles at time of operation. As far as can be felt the ribs have reformed. His only complaint is of a pricking feeling in the scar when he puts his hand to small of back (Fig. 145).

Case 29.—Empyema left (two distinct encysted empyemas). Recovered.

Johan L., twenty-three years. (Episcopal Hospital.)

December 28, 1916. Admitted to the surgical from medical ward with postpneumonic empyema. Temperature, 102°.

Originally admitted to Dr. Deaver's service October 6, 1916, with diagnosis of syphilis, having contracted mixed chancre about one month previously, followed in two weeks by suppurative left inguinal adenitis, there being unhealed sinuses here on admission.

November 6. Transferred to medical ward (Dr. A. A. Stevens) for constitutional treatment for syphilis, local lesions being under control.

December 4. Developed pneumonia at base of left lung.

December 15. Temperature falling by lysis.

December 18. Temperature has not reached normal. Signs of fluid. 100 c.c. of cloudy fluid at second puncture, at angle of scapula. (Culture: Pneumococcus.)

December 21. Flatness extends almost to spine of scapula; heart displaced 2.5 cm. toward right.

December 23. On puncture a small amount of pus recovered from depth at left scapular angle. (Culture: Diplococci.)

December 25. No improvement.

December 27. Temperature slightly higher (101° to 103° since December 24).

December 28. Transferred to surgical ward (Dr. Ashhurst) with diagnosis of empyema.

December 29. In medical ward many weeks with pneumonia, after this temperature reached normal, then rose again and for a couple of weeks has been high and irregular. Five recent punctures negative where signs indicated fluid. Yesterday puncture in eighth interspace below angle of scapula drew 40 c.c. pus (pneumococcus).

Operation. Eucain, 1 per cent. Patient prone. Resected 5 cm. of eighth or ninth left rib and pus oozed before it was removed; pleura nevertheless thick (2 mm.); pus thick, creamy, many curds. Cavity about 8 to 10 cm. in diameter on diaphragm and against costal pleura. Burrowing in adhesions opened *another pocket of pus* above and against ribs. Cavity extended upward toward interlobar region. Lower lobe densely adherent to diaphragm at one point; loose all around. This piece of lung was dissected loose from diaphragm. Considerable complaint and coughing when costal pleura was rubbed in burrowings, but lung and diaphragm insensitive. Drain: Rubber tube and iodoform gauze. No ligatures. No sutures.

January 15, 1917. Drainage lessening.

February 2, 1917. No discharge. Temperature normal. Wound granulating. Up and about ward.

February 9, 1917. Discharged.

February, 1920. The Swedish Consul at Philadelphia reports that this patient was last seen by him about one year ago, apparently in good health.

CASE 30.—Empyema, left. Died after five weeks (sepsis from thoracic wound).

Robert S., seven years.

January 17, 1917. Admitted to the Episcopal Hospital (Dr. Ashhurst's service).

Had had pains in left side and stomach for a week or ten days; January 5, took to his bed; vomited, had cough; chills and high fever. Treated at home for twelve days for bronchopneumonia. Family physician diagnosed empyema following pneumonia.

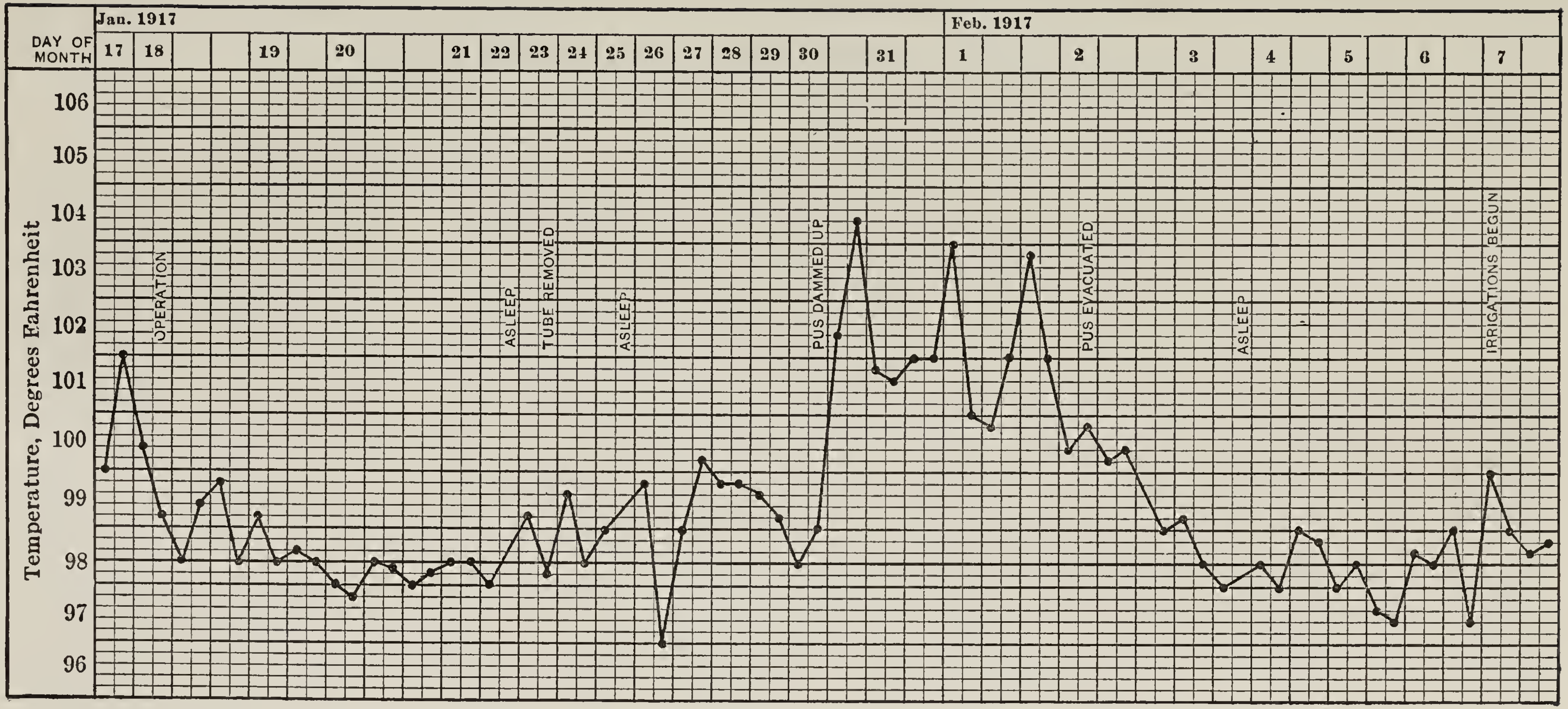

FIG. 146.—Temperature chart of Case XXX, continued as Fig. 147.

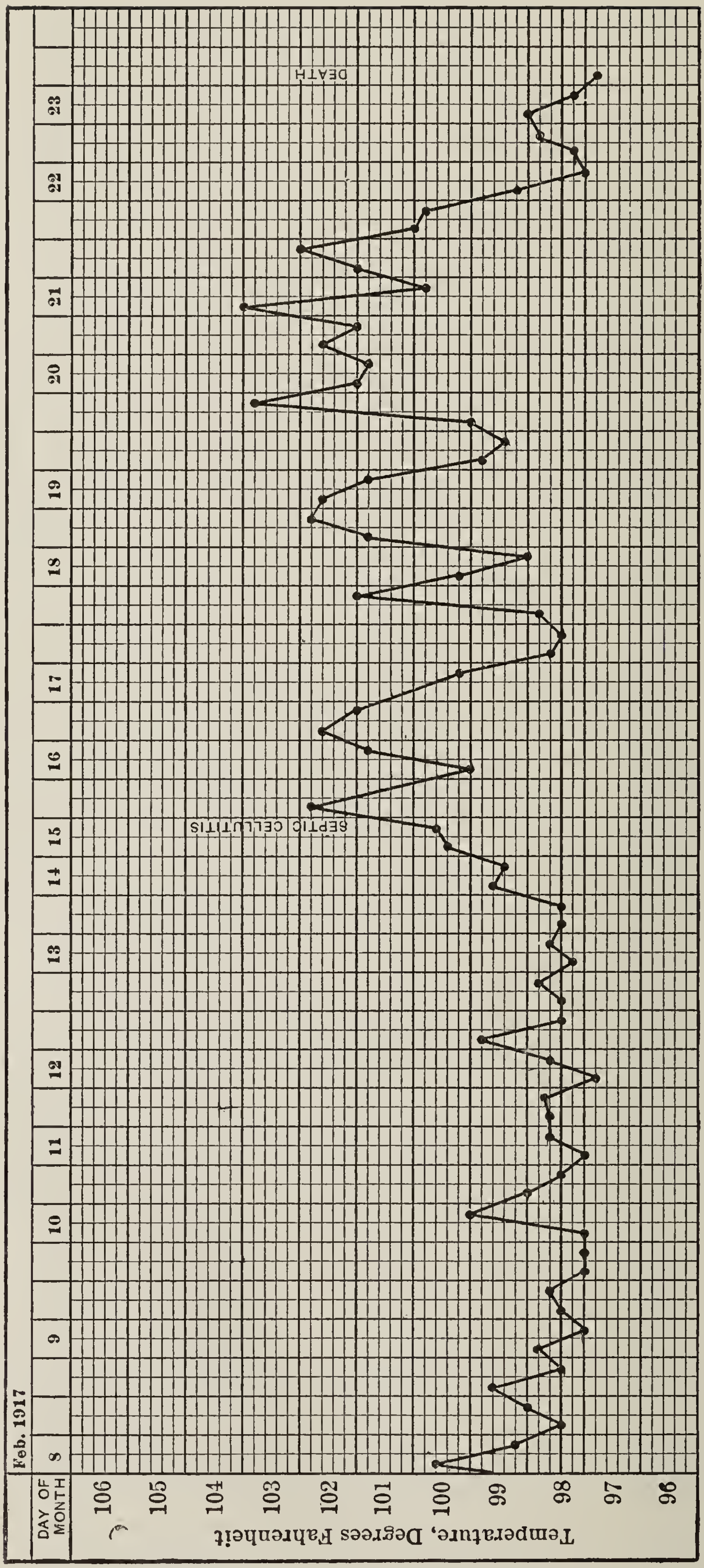

FIG. 147.—Temperature chart of Case XXX. Death from septic cellulitis of thoracic wall. (Continued from Fig. 146.)

January 17. On admission: An anemic, poorly nourished boy. Has incomplete cleft of palate; had operation for hare-lip as infant. Expansion of left chest limited. Palpation negative; dulness and distant breath sounds. *Puncture negative.*

January 18. Operation (Ashhurst). Eucain, 1 per cent. Patient prone. Resected 4 cm. left eleventh rib, posteriorly. Opened pleura, flood of reddish-brown pus. (Culture: Diplococci, pneumococci.) Tube passed upward four inches and held in by silkworm-gut suture. Lung could not be felt. Time fifteen minutes.

January 23. Temperature normal until today. Tube removed. Out of bed.

January 25. Draining slightly. Temperature normal to 100° since removal of tube.

January 26. Up in chair all day; no discharge.

January 27. Wound healing well.

January 31. Temperature rose to 104° suddenly. Transferred to Dr. Mutschler's service.

February 2. Vomits, no drainage. Temperature 100° to 103°.

February 3. Wound explored with hemostat and several ounces of pus obtained. Tube replaced. Temperature fell to normal.

February 5. Draining well.

February 7. In good general condition. Temperature normal. Slight drainage of purulent fluid from tube. Cavity irrigated with normal saline. This causes some pain. Fluid comes back clear. Temperature rose to $100\frac{3}{5}$°. (Fig. 146.)

February 9. Appetite excellent, only slight discharge but child is fretful and nervous. Daily irrigations.

February 12. Temperature, 98° to 100°.

February 15. Temperature rose suddenly to 103° and continued hectic, 100° to 104°, until death, February 23. Daily irrigations.

February 18. Septic temperature, with sweats. No chills. Vomited. Unproductive cough. Small area of cellulitus around drainage tract, with extreme tenderness. Chest negative but no pus draining. Pulse-rate now 120 to 140, formerly 80. Respiration 28 to 32, formerly 20. Slightly impaired percussion note over left chest posteriorly; two punctures negative. Irrigations continued.

February 20. No drainage. Unable to sit up without support; will not eat. Vomits at intervals. A maculopapular rash like measles over trunk and extremities. Throat negative. Irrigations continued.

February 21. Sweats, paroxysms of vomiting without other abdominal signs or symptoms, anorexia and increasing weakness continue.

February 22. Irrigated. Tract is open but there is no drainage. Marked abdominal distention but no pain or rigidity. Rash still present. Temperature falling.

February 23. Died with acute dilatation of heart 11.30 A.M. Temperature subnormal. Cause of death: Sepsis from wound of thoracic wall.

CASE 31.—Empyema right. Died (sepsis from thoracic wound).

Stanley K., nine years.

January 15, 1917. Admitted to the Episcopal Hospital, Dr. Ashhurst's service. Present illness began on December 24, 1916, with chill, fever, vomiting and jaundice. Four days later pain in right side, radiating to shoulder, and to left chest, finally localized to right costal margin. Short of breath and breathing pained him. Was treated for lobar pneumonia. Two days before admission mother noticed right chest was swollen; called doctor's attention to it and he found pus on puncture.

On admission, right lower chest had interspaces obliterated, flat, absent breath sounds, apparently massive empyema. Two punctures in axilla negative, but pus found below angle of scapula (Dr. Spruance, ward surgeon); 200 c.c. pus removed by puncture. Smear and culture negative. Polynuclears, 55 per cent. Lymphocysts, 45 per cent. Temperature, $101\frac{3}{5}$° F. Pulse, 160. Respiration, 48.

January 16. Operation (Ashhurst) (eucain, 1 per cent.). Patient prone. Resected 4 cm. of 11th right rib. Pleura opened just above its reflection on to diaphragm. No pus found here. Inserted finger and in adhesions above 10th rib, close to spine, got a gush of pus. (Culture: Negative.) Pus reddish-brown, no curds. Cavity runs up spinal gutter as far and as wide as finger can reach. Inserted rubber tube upward 10 cm. and more pus then spurted when he coughed (1 liter in all).

January 21. Draining considerably.

January 22. Tube removed.

January 25. Still draining. Out of bed on house diet. Temperature, 99° to 100°.

January 26 to 31. Temperature, 99° to 103°. Pus probably dammed up.

January 31. Transferred to Dr. Mutschler's service.

February 1, 1917. Wound closed. Temperature irregular.

February 1 to 5. Temperature, 98° to 103° F.

February 7. Operation wound granulating, no discharge. On deep inspiration (which brings on coughing) there is limitation of expansion on right side above. Tactile fremitus absent and breath sounds distant in right scapular region. At right base many moist bubbling rales. Percussion note hyperresonant except in scapular region, where it is flat.

February 6 to 8. Temperature, 99° to 102°.

February 8. Finger inserted in old sinus (no anesthetic); base of lung felt to crepitate and expand with inspiration. Adhesions bound an area from 9th rib above, in front by posterior axillary line, below by diaphragm. These adhesions cannot be broken by gentle pressure and more is not attempted because of fear of pleural reflex. Temperature fell from 102° to 99°.

February 10. Dyspnea increasing. Drenching sweats twice daily. Well-defined area of flatness extending from scapular spine to its angle and as far forward as midaxilla. Puncture in 6th interspace at angle of scapula and creamy purulent fluid is withdrawn about the consistency of molasses in March. A second futile attempt is made to drain this encysted collection of pus through the old incision.

February 9 to 12. Temperature, 99° to 101° F.

February 12. General condition excellent.

February 13. Operation (Mutschler) (ether); 3 cm. of 7th right rib resected in midscapular line and 2 ounces (60 c.c.) of creamy, purulent, viscid fluid evacuated. Pleural cavity is then irrigated with sterile water until latter comes away clear. Rubber tube inserted 6 cm., stitched to skin by silkworm-gut. Incision is then closed with interrupted silkworm-gut sutures and dressings applied. On return to ward was able to breathe comfortably in recumbent position.

February 14. Dressings soaked with purulent blood-stained discharge. Old tract has definitely closed. Pleural cavity is irrigated with warm normal saline solution, injected through rubber drain tube and again withdrawn.

February 15. Dressings and irrigation repeated. Unable to sit up without excruciating pain in right scapular region. The wound is draining well, but temperature continues elevated (100° to 103°).

February 17. Drainage less. Well-defined area of induration, redness, tenderness, swelling and edema around drainage tube. Irrigation is followed by a fit of coughing and fluid expelled is seen to contain small pieces of coagulated exudate.

February 17 to 20. Temperature, 100° to 104°, and so on until death, February 25.

February 19. Pulse feeble, rapid but regular. Vomited. Takes liquids sparingly, no food. Macular rash, coppery in color, over limbs and trunk. Is evidently exceedingly toxic; wound drains little. Rubber tube removed, irrigation performed. Percussion note is resonant to level of 9th rib; below this there is flatness, with distant breath sounds. Puncture in 9th interspace causes no pain; clear, colorless fluid, evidently residue of irrigating fluid, is withdrawn; contains small fragments of floating exudate.

February 20. Obviously very ill. No appetite, listless all day long. Area of cellulitis around tube is larger. Movements are painful, as if they involve the muscles in this area. Sutures removed.

February 24. Vomited large quantity of greenish material. Dyspneic, toxic, dull and drowsy. Toward evening cyanotic.

February 25. Died 3.30 A.M. Cause of death: Sepsis from wound in thoracic wall.

Case 32.—Empyema right. Recovered.

Whitman L., forty-six years.

January 3, 1917. Admitted to the Episcopal Hospital (Dr. Stevens's service). Has had a cough for years. Last evening was taken with chills and sweats, vomiting and severe headache. This morning had general pains and pain on inspiration in right chest. Typical pneumonia at right base.

January 12. Signs of fluid at right base. Temperature has never reached normal (99° to 101°).

January 14. Developed bilateral suppurative otitis media. Paracentesis of right chest negative.

January 17. Temperature rising (101° to 103°). Still signs of fluid at right base. Is not septic.

January 22. Signs of fluid at right base; puncture gave pus. (Culture and smear: Diplobacillus.)

January 23. Puncture draws 250 c.c. creamy pus.

January 24. Transferred to surgical service (Dr. Ashhurst). Operation by Dr. Ashhurst (eucain, 1 per cent.). Patient prone. Resected 4 cm. of eleventh rib; found very much thickened pleura, cut through it and exposed fibers of diaphragm. Then cut higher in pleura (costophrenic sinus) and opened pleural cavity, whence over 500 c.c. of pus was discharged; creamy, inodorous. (Culture: Diplococcus and pneumococcus(?)). Diaphragm not sensitive, costal pleura very sensitive. Cavity extends up and back beyond reach of finger. Drain: Large rubber tube up for 10 to 12 cm.

January 26. Drainage almost stopped. Sat up in chair a while.

January 31. Still draining profusely. Temperature normal ever since operation.

February 10. Less drainage. Learning to walk.

February 12. Tube removed.

February 17. Went home.

February 29, 1920. Wound healed firmly four weeks after leaving hospital. No disability. Lungs normal.

Case 33.—Empyema, right. Recovered.

John B., twenty-six years.

January 10, 1917. Admitted to the Episcopal Hospital, Dr. Stevens's service. Illness began January 4 with chill and sweat, and pain in right side; has had sweat almost every night. On admission: Exceedingly toxic and sick, entire body deeply jaundiced. Cyanosis of finger-tips. Pneumonia at base right lung.

January 12. Has been markedly delirious. Strapped to bed. Temperature low, never up to 101°, evidently poor reaction.

January 16. Rational. Jaundice subsiding.

January 23. Signs of fluid at right base (apical pneumonia). Puncture gives pus. Temperature 101° to 103° since January 21.

January 24. Transferred to surgical ward (Dr. Ashhurst).

Operation by Dr. Ashhurst (eucain, 1 per cent.). Patient prone. Resected 4 cm. of 11th rib; pleura thickened, pus gushed on opening it. Finger introduced, found opening at lowest limit of cavity, which extended up and back beyond reach of finger, limited in front and above by attachments of diaphragm. About 1 liter of curdy yellow pus evacuated (Culture: Pneumococcus), and continues to drain profusely when tube is inserted, up 10 to 12 cm. along spine.

January 26. Drainage almost stopped. Sat up in chair a while.

January 31. Still draining profusely.

February 6. Sits up each day. Temperature, 98° to 102°.

February 12. Tube removed, temperature having reached normal.

February 18. Discharged. Temperature normal since last note. Wound nearly healed. Patient not traced.

Case 34.—Empyema, right. Recovered.

William D., sixteen years.

January 9, 1917. Admitted to the Episcopal Hospital, Dr. Stevens's service. Chief complaint: pain in right chest worse on coughing and on deep inspiration. Temperature, 103°. Onset January 5, with chill and sweat; cough; blood-stained sputa; headache and weakness. Excessively ill with typical lobar pneumonia, right middle lobe.

January 11. Type II pneumococcus reported in sputum.

January 12. Pericardial friction sounds. Very toxic. Abdomen markedly distended. Temperature fell by crisis to 99°.

January 16. Temperature has gradually risen again to 103° F. Pneumonia at *left* base.

January 21. Still very ill. Temperature irregular, 100° to 103°. Sweats at night. Signs suggestive of fluid at right base. Consolidation at left base.

January 25. 250 c.c. pus drawn by puncture in ninth interspace, posterior, right side.

January 26. Transferred to surgical service (Dr. Ashhurst). Temperature fell to normal after puncture. Is blue, thin, anemic. Right chest (lower) is flat, distant breath sounds, somewhat diminished fremitus.

Operation by Dr. Ashhurst (eucain, 1 per cent., 10 c.c. only). Patient prone. Resected 4 cm. of eleventh right rib; pus oozed before rib was resected. About 500 c.c. creamy yellow pus evacuated. Culture: Diplococcus (pneumococcus). Finger introduced, felt no lung or adhesions. Opening was at bottom of costophrenic sinus. Large rubber tube passed up 8 to 10 cm. and held to upper skin margin by silkworm-gut suture. Time ten minutes.

January 27. Temperature normal.

January 31. Temperature irregular, 98° to 102°.

February 3. Still draining freely.

February 6. Out of bed daily. Temperature still irregular.

February 12. Smaller tube inserted.

February 18. Temperature normal. Free drainage. Learning to walk.

February 19. Tube removed.

February 21. Wound closes between daily dressings, damming up a few drops of pus.

February 22. Small tube reinserted. Went home.

February 21, 1920. Three years after operation. Scar has remained healed since discharge from hospital. Then weight was 87 pounds. In four weeks it reached 114 pounds and is now steady at 120 pounds. Went to State Sanatorium for Tuberculosis on discharge and remained there for nearly a year. Lungs are normal on examination, except slightly distant breath sounds over right chest posteriorly. Chest expands normally. Well-developed chest. No deformity. Rib has reformed. Some evidence of old endocarditis audible and gets out of breath easily, but able to do a man's work.

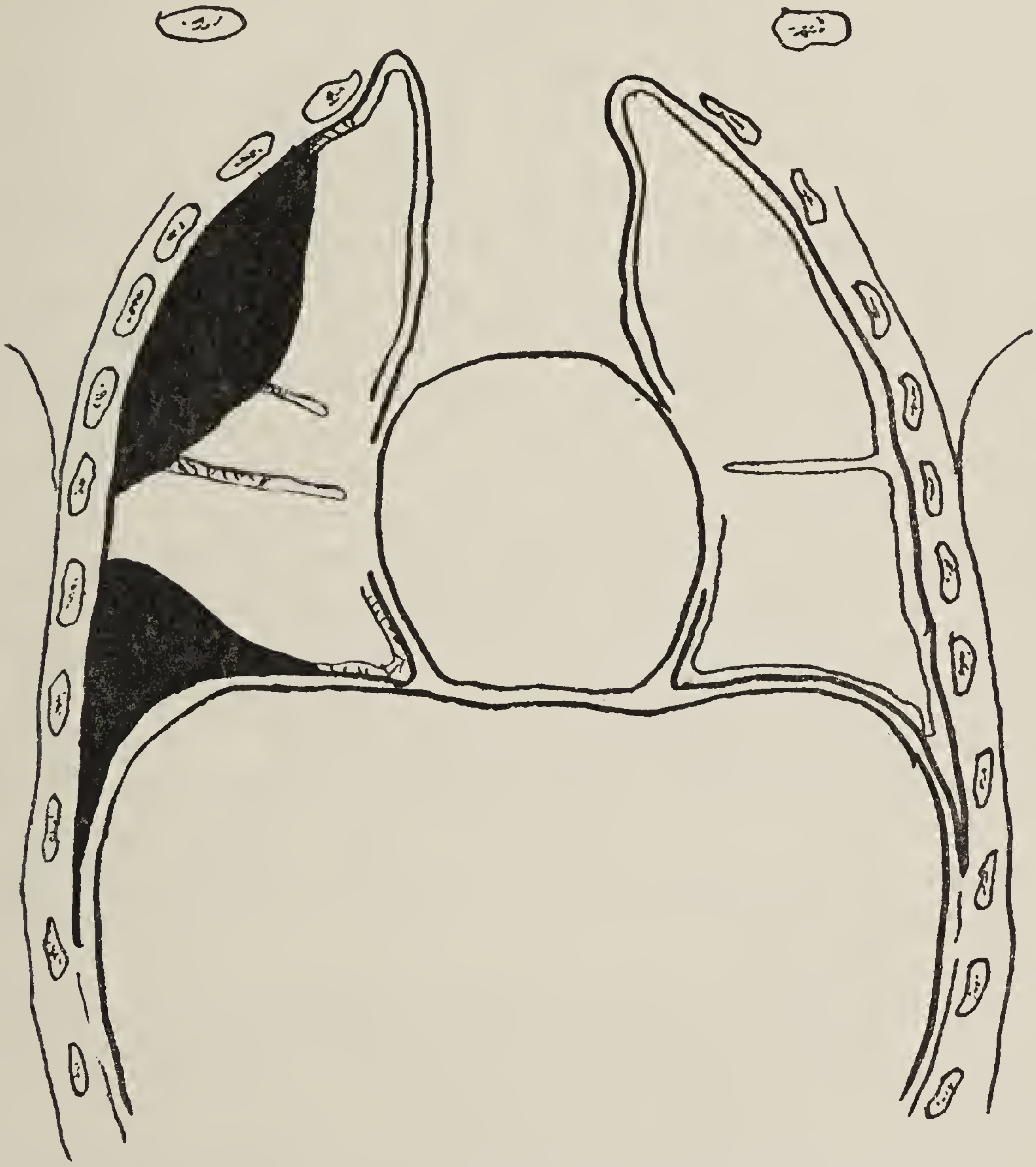

Fig. 148.—Case XXXV. Two distinct encapsulated empyemata.

Case 35.—Empyema, right (two distinct encysted empyemas). Recovered.

Edward H., thirty-two years.

April 7, 1917. Admitted to the Episcopal Hospital, Dr. Ashhurst's service. Referred by Dr. H. R. M. Landis.

Taken ill ten weeks ago. Diagnosis: Pneumonia, right. Sent to a hospital and after about seven weeks, as he did not get well, they said he was tuberculous (called it "unresolved pneumonia") and sent him to White Haven, Pa. Seen there March 17, 1917 (three weeks ago), by Dr. Landis, who drew off a quantity of *sterile* pus, and referred him to Dr. Ashhurst.

Examination: Emaciated, free expectoration of mucopus (not fetid). Right chest dull over lower lobe and signs of fluid. Needle drew from 8th interspace in posterior axillary line, 350 c.c. of pus (92 per cent. polynuclears. Smear: Negative).

April 8. Operation by Dr. Ashhurst (eucain, 1 per cent.). Resected 5 cm. of 9th right rib (patient prone). The pleura much thickened (2 to 3 mm.). (Spurting intercostal artery tied by suture, caused much pain, checked by eucain injection.) On opening pleura pus escaped from *thoroughly walled-off abscess between diaphragm and lung.* Diaphragm felt normal, soft, depressible; and smooth liver palpable beneath it. The upper margin of cavity was formed by lung, densely adherent at periphery to costal pleura, just above 9th rib. Diaphragm rose to 10th rib. Supraphrenic abscess extended four inches toward dome of diaphragm. With much difficulty, but *without causing any pain,* adhesions of lung to costal pleura were broken through by the fingers, first far posteriorly, and then the lung was peeled off the costal pleura all the way front (here it was painful, under anterior costal margin, 8th, 7th and 6th ribs).

It was noted that the needle puncture which drew pus in the 8th interspace was above the level where the lung was adherent to the chest wall. *On detaching the lung a perfect flood of pus (over* 500 *c.c.) came from above, from between lung and ribs;* this cavity extended up as far as finger could reach. (Culture: Diplococcus.) (Fig. 148.)

Drain: Rubber tube between lung and diaphragm and another (2 cm. lumen) between lung and costal pleura, running up toward apex 15 cm. Iodoform gauze stuffed in wound between these two tubes to hold margin of consolidated lung away from wound and convert the two abscess cavities into one. Wound not sutured (12.5 cm. incision). Time, thirty minutes.

May 27. Uneventful convalescence. Wound has been merely moist for last two weeks.

June 3. Went home. Wound now closed for a week with normal temperature, though up to then small tube entered six inches easily. Weight, 150 pounds; gain of 23 pounds in eight weeks since operation. Breath sounds normal. No symptoms. Right chest moderately contracted in front.

July 6. Reports he will return to work in one week.

November, 1917. Sinus discharged but healed in three weeks.

February, 1919. Empyema formed again and another operation, 5 cm. above former operation, done by Dr. Schell at Northwestern General Hospital. Recovered. Weight, 170 pounds.

September, 1919. Moved to Denver. Since then one reaccumulation was opened; healed in six weeks.

March, 1920. Weight, 170 pounds. Excellent health, no symptoms of tuberculosis; working every day.

Case 36.—Empyema, right; encapsulated. Recovered.

Oliver C., twenty-one years.

July 9, 1919. Admitted to the Episcopal Hospital, Dr. Robertson's service. Ill since July 7; typical pneumonia. Chief complaint is pain in right chest and shortness of breath. On awakening in morning had chill, became feverish and dyspneic and developed pain in right chest on inspiration. On admission: Signs of pleuropneumonia at right base. Temperature, 100° to 102°. Thought to have fluid, but puncture negative.

July 12. Puncture in posterior axillary line is negative.

July 14. Consolidation also of left base.

July 15. Pleuropericardial friction sounds on left.

July 24. Temperature continues 99° to 103°.

July 25. Temperature declined by lysis, yesterday shot up again to 103°. Needle in 9th right interspace in scapular line drew 350 c.c. pus. (Culture: Pneumococci.) Transferred to surgical service (Dr. Ashhurst) July 26.

Operation by Dr. Ashhurst (novocain, $\frac{1}{4}$ per cent.). Excision of 5 cm. of 10th rib. Costal pleura normal but adhesions of lung to costal pleura above and in front of opening, and lung adherent to diaphragm. Could not be separated by fingers. Packed. Lung cut loose from diaphragm by scissors. 150 c.c. pus, yellow, creamy. Tube and gauze wick. A few whiffs of ether toward end. Diaphragm felt normal for 5 cm. from ribs, then felt hard; this part was adherent to lung.

August 3. Temperature has reached normal. Little discharge.

August 13. Tube removed. Has been up in chair several days and rapidly getting fat.

August 14. Temperature shot up to 103°. Tube replaced.

August 18. Last night and night before had chills. Resident's attempts to get tube in far enough apparently ineffectual. Therefore today, under gas, Dr. Ashhurst put finger into sinus and found lung again adherent to diaphragm, and tore it loose, giving exit to bloody pus; and put tube again deep in wound, almost to dome of diaphragm.

August 25. Temperature has gradually reached normal. No discharge.

September 5. Tube removed. No discharge.

September 6. Temperature shot up to 103°. Tube replaced.

September 8. Temperature normal.

September 14. Tube removed.

September 15. Temperature shot up to 102°. Tube replaced.

September 17. Temperature normal. No discharge.

September 20. Out of bed.

September 27. Temperature stays normal, but sinus does not heal, though there is practically no discharge. Tube retained.

October 10. Tube shortened.

October 15. Tube removed and gauze wick inserted.

October 21. Bismuth paste injected to determine size of empyema cavity. Roentgen ray shows sinus extends up to 5th rib anteriorly. (Roentgen ray 1224 D.)

October 25. No discharge from wound since injection of bismuth.

October 30. Wound is firmly healed. Went home.

November 17. Returns on visit. Still healed. No symptoms.

February 29, 1920. Weight 170 pounds (127 pounds on leaving hospital). Went to work the day after discharge from ward, and has been at work ever since. No disability. Lung normal except distant breath sounds at right base posteriorly. The rib has not yet reformed.

Case 37.—Empyema, right (massive). Recovered.

Isador M., eleven years.

August 4, 1919. Admitted to the Episcopal Hospital, Dr. Robertson's service.

Onset July 31. On admission very ill, lobar pneumonia at right apex.

August 6. Puncture at angle of scapula gives 12 c.c. turbid fluid.

August 8. Puncture in axilla through 4th interspace and at angle of scapula in 8th interspace; 3 c.c. of slightly turbid pus obtained from each. Patient is better.

August 9. Puncture just lateral to angle of scapula gave 500 c.c. thick green pus. Much better after evacuation.

August 12. Puncture gives 350 c.c. of green pus. Temperature declining by lysis. Smear shows diplococci of pneumococcus type.

August 14. Temperature 98° to 102°.

August 16. Puncture gives 250 c.c. of pus. Very thick and green; 20 c.c. of formalin in glycerin injected. Temperature fell from 103° to 99°.

August 18. No change. Examination by Dr. Ashhurst. Skodaic resonance over right apex anteriorly; interspaces on right side of chest obliterated. Dull in axilla and posterior to line of nipple, and posteriorly dull below the 4th interspace except close to vertebral column. Breath sounds distant. Diagnosis: Postpneumonic empyema. Duration ten days. Transferred to surgical service (Dr. Ashhurst).

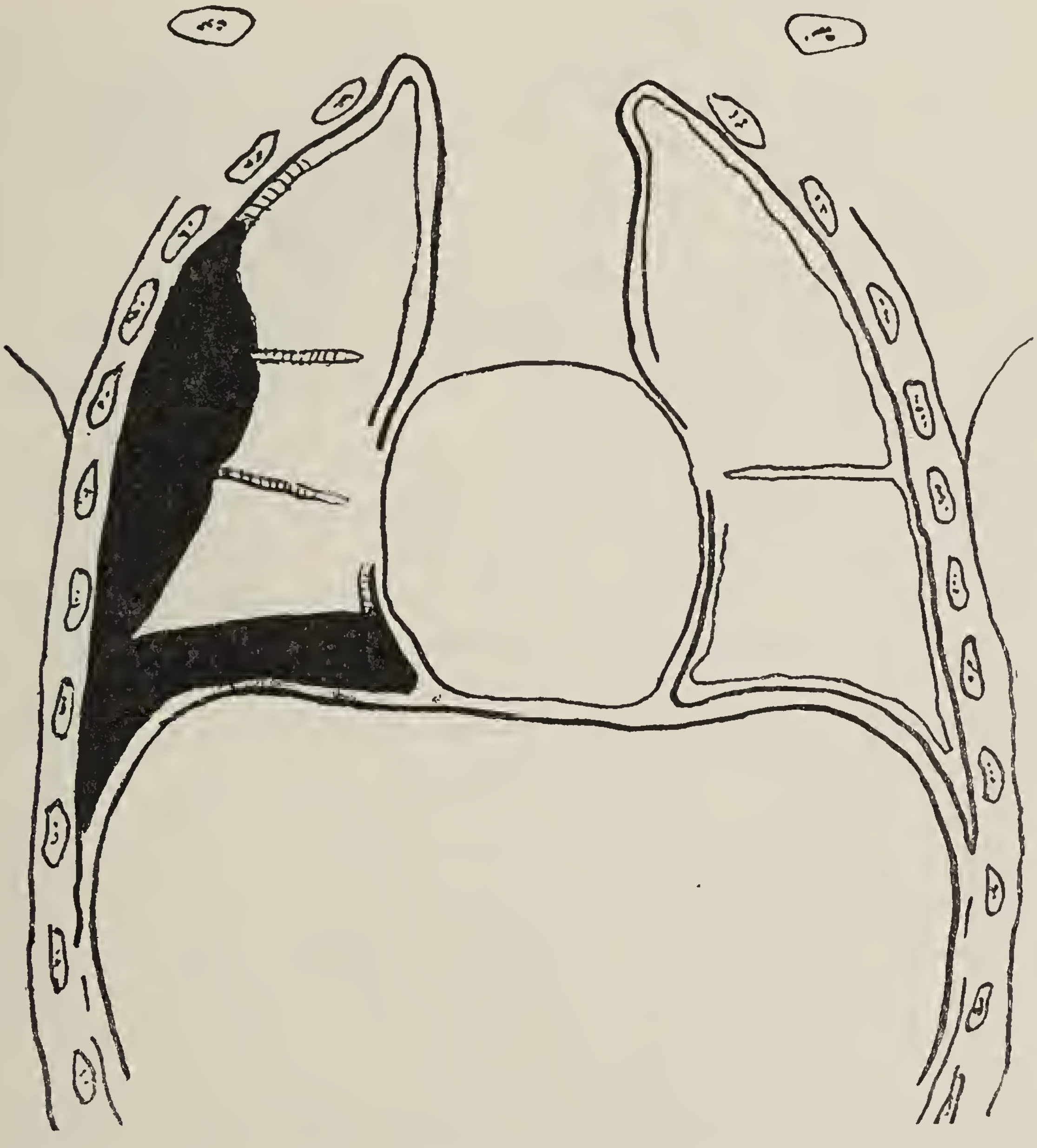

FIG. 149.—Case XXXVII. Massive hour-glass empyema. Case XLIII was quite similar.

Operation by Dr. Ashhurst (novocain, ¼ per cent.). Resection of 4 cm. of 10th right rib posteriorly. One liter of pus (Culture: Pneumococci) evacuated on opening pleura. The cavity extended from diaphragm as high as finger could reach and also between diaphragm and lung (Fig. 149). Diaphragm was covered with thick, gray slough, and the lower lobe of the lung stood out surrounded by pus on both phrenic and costal surfaces.

August 22. Free drainage. Doing very well.

September 1. Wound closing. Less discharge.

September 9. Tube removed.

September 13. Incision healed. Sent home.

March 21, 1920. Seven months after operation. Has gained 14 pounds in weight since leaving the hospital—present weight 83 pounds. Lungs are normal. Rib appears to have reformed.

Case 38.—Empyema, right. Recovered.

Evelyn S., nineteen years.

November 16, 1919. Admitted to the Episcopal Hospital, Dr. Ashhurst's service. Sent from Philadelphia Hospital for Contagious Diseases, where she was taken October 31 for suspected diphtheria. October 29. Onset with sore-throat and headache. October 31. Taken to Hospital for Contagious Diseases. November 5. Pain in right side. Dulness. November 13. Evidences of fluid in right chest. Puncture drew 20 c.c. yellowish purulent fluid (sterile). Throat has given nine negative cultures, never a positive culture.

On admission, chief complaint is pain in both arms and back. There is a subpectoral abscess below right clavicle. Patient emaciated and very septic. Temperature, 103° F.

November 17. Seen by Dr. Fussell, who advised aspiration over right base, where signs were suggestive of fluid.

November 18. Dulness and distant breath sounds over lower posterior chest. Puncture draws pus. Diagnosis: Right empyema. Operation by Dr. Ashhurst (novocain, $\frac{1}{4}$ per cent.). Resection of 3 cm. of 9th rib, below angle of scapula. Pleura thickened. 250 c.c. of greenish-yellow, creamy, malodorous pus evacuated (same as by aspiration). (Culture: no growth. Smear: organisms like streptococci but smaller.) Cavity extends backward to spine and upward beyond reach of finger. Rubber tube sutured in wound. Right subpectoral abscess to be treated later. Diaphragm came just to level of rib resection and felt flat across top to spine; whole cavity appears rigid and may require secondary operation for closure. Still very ill.

November 19. Had a chill.

November 20. Improving, free drainage of pus.

November 22. More drainage than usual from tube. Subpectoral abscess much smaller. Still very annoying cough.

November 25. *Second operation.* Large abscess from beneath left scapula drained under local anesthesia. (Smear: Diplococci and staphylococci.) First sign of this abscess developed seventy-two hours previously. Still very ill.

November 30. The subpectoral abscess has refilled.

Third operation: 100 c.c. of pus from beneath right pectoralis major drained under local anesthesia. (Smear: Diplococcus and bacillus. Culture: no growth.)

December 1. Better. Still coughing. Incision in left back almost healed.

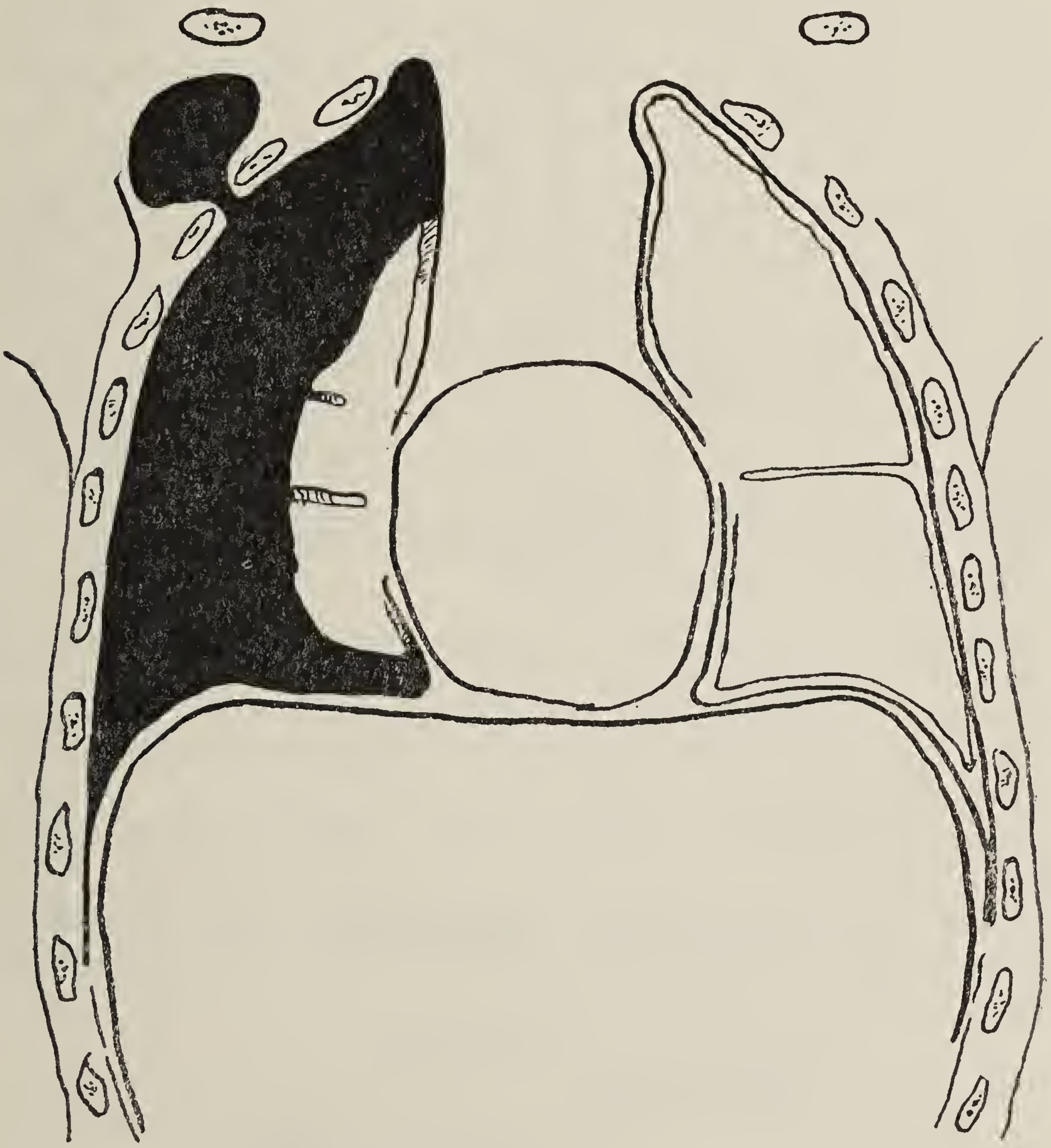

FIG. 150.—Case XXXVIII. Massive empyema necessitatis, pointing beneath right pectoral muscles. With metastatic abscess between left scapula and chest wall.

December 20. Out of bed in wheel chair.

January 9, 1920. Tonsils removed under local anesthesia.

January 28. All incisions now firmly healed, the last to close was that of subpectoral abscess. Empyema incision has been healed for two weeks. Went home today.

March 21, 1920. Four months after operation, incision has remained closed. Is round shouldered, with slight scoliosis convex to the left. Gets tired easily.

Case 39.—Empyema, right. Recovered.

Gertrude W., four years.

December 5, 1919. Admitted to the Episcopal Hospital, Dr. Ashhurst's service.

Had whooping-cough and influenza in fall of 1919. Four weeks ago she began vomiting; physician made a diagnosis of congestion of lungs. Later he diagnosed pleurisy.

On admission: Anemic, run-down child, with respiratory embarrassment. Respiratory movements almost absent on right. There is markedly diminished tactile and vocal fremitus of whole chest, anteriorly and posteriorly. Percussion shows flatness over entire right chest. Puncture draws 75 c.c. of thick greenish pus.

December 6. Operation (Dr. Moore, ward surgeon). Novocain, 1 per cent. Resection of 3 cm. of right 9th rib below angle of scapula. 100 c.c. pus evacuated. (Smear: Diplococci and staphylococci. Culture: Mixed.)

December 24. Rise in temperature. Longer tube inserted.

December 27. Temperature down.

December 29. Tube removed.

January 7, 1920. Rise of temperature for past three days; 30 c.c. pus evacuated by forceps in sinus. Tube replaced.

January 13. Temperature normal since replacing tube. Tube removed today.

January 22. Out of bed for four days. Sent home with incision healed.

April 11. In good health. Lungs normal. Four weeks after leaving hospital a little pus discharged from sinus on removing scab. Healed in three or four days.

Case 40.—Empyema, left. Recovered.

Thomas W., sixteen months.

December 31, 1919. Admitted to the Episcopal Hospital, Dr. Carson's service.

Eleven days ago child developed fever and cough; physician called who diagnosed pneumonia. After convalescence fever recurred and physician suspected empyema. On admission there is limited expansion of left chest, no fremitus on palpation and dulness over entire chest continuous with heart dulness. Hyperresonance over right lung. Heart displaced 2 cm. to right. Very slight change in breath sounds on left side; on the right side breath sounds are clear and distinct. Has a cough.

Puncture of left pleura gives 100 c.c. turbid straw-colored fluid, no odor (smear and culture show diplococci).

January 2, 1920. Not sleeping well; dyspneic. Pulse not so good. Puncture gives 250 c.c. turbid odorless greenish straw-colored fluid; some relief of dyspnea.

January 4. Slightly better. Fluid collecting again.

January 7. Puncture gives 150 c.c. greenish-yellow turbid fluid, no odor. Gradually improving.

January 8. Restless and dyspneic. Very irritable. Seen by Dr. Ashhurst in consultation and transferred to his surgical service.

January 9. Operation by Dr. Ashhurst (novocain, $\frac{1}{4}$ per cent.). Resection of 2 cm. of 10th left rib, midscapular line. Pleura 25 mm. thick. Thin, serous pus evacuated. Cavity extends as far as finger can reach. Tube drainage.

January 12. Temperature normal since yesterday.

January 14. Tube removed. No pus has drained since operation.

January 29. Temperature rose to 104° yesterday, after being nearly normal since last note; 10 c.c. of pus evacuated from sinus by inserting forceps. Then temperature fell to normal. Tube replaced.

January 31. Transferred to Dr. Neilson's service.

February 19. Temperature has been normal for four days, after another flare-up, relieved by evacuating a little pus.

February 25 to March 9. Temperature, 100° to 102° F.

March 9. Not doing well. Septic temperature. Nothing definite in chest findings, but roentgen-ray examination indicates presence of pus. Under ether anesthesia, Dr. Hawfield (chief resident physician) ran finger into sinus but found no pus. Tube left in sinus.

March 12. Free discharge of pus.

March 20. Temperature normal for past week.

March 26. Sent home, with tube still in sinus.

April 2. Tube out. Tiny sinus.

April 16. Wound healed. In perfect health.

Case 41.—Empyema right. Recovered.

Alice M., twenty-nine years.

February 6, 1920. Admitted to the Episcopal Hospital (service of Dr. J. B. Carson).

Chief complaint: pleurisy pains. Eight days ago was taken suddenly with chill and feverishness. Some cough. Three days ago severe pain in right chest; constant since that time.

On admission: Consolidation, base right lung with Skodaic resonance above.

February 8. Puncture of right chest gives 250 c.c. serous fluid. (Smear shows a streptodiplococcus probably pneumococcus; culture is contaminated.)

February 10. Condition fair. Temperature falling by lysis.

February 18. Temperature since February 12 has been rising gradually and since 14th has been 100° to 103°. Transferred to surgical service of Dr. Mutschler.

February 20. Operation (Dr. Ashhurst). Novocain, $\frac{1}{4}$ per cent. Patient prone. Resection of 4 cm. of 11th right rib. Nearly 500 c.c. of creamy yellow pus evacuated upon opening pleura, which was 2 or 3 mm. thick. (Culture: Pure pneumococci.) Finger introduced palpates no lung as high as it can reach. Opening is at level of diaphragm, which is flat, not domed as normally. Large tube drainage.

March 5. Tube replaced by smaller one. Very little discharge; up and about ward. Temperature has slowly fallen to normal.

March 10. Up and about ward. Very little discharge.

March 30. Tube removed.

April 12. Wound healed.

April 15. Went home.

June, 1920. In good health.

CASE 42. Empyema, right. Recovered.

Theresa D., twenty-seven years.

February 7, 1920. Admitted to the Episcopal Hospital, Dr. J. B. Carson's service. Chief complaint: cough, backache and pain in the chest. Husband has pneumonia and three children have influenza. Patient has been sick in bed four days. Has a nursing baby.

Examination: Limitation of expansion and impaired resonance over *left* chest.

February 11. Improved.

February 14. Not so well.

February 15. Consolidation at *right* base.

February 18. Signs of fluid at right base. Two punctures negative; third puncture found sero-pus. (Culture: Pneumococci.) Transferred to surgical service of Dr. Mutschler.

February 20. Examination by Dr. Ashhurst. Entire right chest posteriorly is flat from spine of scapula down to base, and breath sounds are distant.

Operation by Dr. Ashhurst (novocain, $\frac{1}{4}$ per cent.). Patient prone. Resection of 4 cm. of 10th right rib near its angle. Pleura slightly thickened. Upon opening it, thin turbid pus discharges, with some flakes of lymph, nearly 500 c.c. Finger introduced feels expanding lung, not bound down by adhesions. Large tube drain.

March 5. Tube replaced by smaller and shorter. Temperature normal ever since operation.

March 19. Up and about ward. Very little discharge.

April 2. Tube removed.

April 12. Wound healed.

April 15. Went home.

CASE 43.—Empyema, left.

James S., thirty-four years.

April 7, 1920. Admitted to the Episcopal Hospital, Dr. Hopkins's service. Onset February 6 with influenza, followed by pneumonia with crisis in eight days. Did not get well. Thought by family physician to have tuberculosis. Two aspirations at home four weeks ago in posterior axillary line left side were negative.

April 8. Aspiration showed thick creamy pus. Aspirated in 7th interspace, posterior axillary line. Transferred to surgical service (Dr. Ashhurst). Emaciated, septic adult, with beginning bed-sore over sacrum. Has constant cough.

April 9. Operation (Dr. Ashhurst). Novocain. Resection of 4 cm. of 10th rib in scapular line. Pleura thickened. Pus spurted 2 meters on opening pleura, creamy, greenish-yellow pus. About 2 liters removed. Exudate surrounding lung on costal and diaphragmatic surfaces (Fig. 149). Tube inserted and anchored with silkworm gut.

April 10. Coughed very little.

April 19. Tube removed and replaced by smaller. Very little discharge at any time since operation. Uninterrupted convalescence.

April 21. Out of bed.

April 23. Tube shortened.

April 30. Smaller tube inserted.

May 12. Tube removed.

May 15. Went home; granulating wound; no discharge.

May 25. Wound healed.

August, 1920. Four months after operation. In perfect health; lung normal.

A REPORT OF THIRTY CASES OF EMPYEMA FOLLOWING INFLUENZAL PNEUMONIA

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AND

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Following the two recent influenza epidemics there were 65 cases of empyema treated in this hospital. Of this number practically all gave a history of influenza followed by a frank pneumonia. It is of interest to note that the empyema cases of the first epidemic were in greater proportion than during the more recent epidemic, probably because of the apparently more severe infection of the first epidemic. Then too, the pneumonia patients of the first epidemic could not receive the attention that should have been given them owing to the great shortage of physicians during the recent war. A good many patients who entered in the hospital during the first epidemic died shortly after admission and were found to be cases that had not received proper attention at home.

Following and during the first epidemic, there were 47 cases, and in the more recent epidemic there were 18 cases. Of the 47 cases during the first epidemic 30 were on the service of Dr. H. C. Deaver, and it is the purpose of this paper to discuss briefly (1) the diagnosis and etiology; (2) to outline the forms of treatment, and (3) to discuss the clinical course of these 30 cases.

Diagnosis. The diagnosis in most instances of empyema is an easy matter, for in a great majority of cases there is a definite clinical history, definite physical signs and *x*-ray findings, and on aspiration one is usually able to locate the pus. Of course there are cases of encysted fluid or pus, and in such cases the diagnosis is not always an easy matter, but careful exploratory puncture and observation of the symptoms and signs together with the fluoroscopic and *x*-ray examination of the chest usually enable one to find the fluid.

The early diagnosis of empyema is of great importance, and yet, as will be pointed out later, immediate operation is often contra-indicated, for the patient's physical condition is often such that less drastic means should be undertaken until such time as it is safe to operate. Just as in inflammatory processes in the abdomen, Nature should be given a chance to establish her barriers in a great many cases of empyema. It was shown during the war that in those cases in which early operation was done the mortality was heavier than in those where there was delay sufficient to allow nature to wall off the inflammatory process. Sufficient time should be allowed to elapse before operation to allow resolution of the pneumonia process to take place.

Etiology. The etiology of empyema in most cases is secondary, and when occurring in the course of pneumonia it is very probably nothing more at first than a small subpleural abscess, which later breaks through the pleura, setting up either a local or a diffuse inflammation of the pleural lining. The causative organism in practically every case reported in this article was the pneumococcus, but in most cases a mixed infection was present. The influenza bacillus was not recovered in a single instance.

It was observed during the more recent epidemic of 1920 that the majority of cases began as a serous effusion, for in early aspiration only a clear serous fluid was obtained, and later with a continuation of the clinical symptoms a second aspiration produced a thick, creamy looking pus and culture of this revealed a pure culture of pneumococcus. The pus-producing organisms are present probably from the first but do not produce an increase in the leukocytes at first sufficient to give the exudate a typical purulent appearance. During this early stage, aspiration should be done only to relieve the circulatory and respiratory embarrassment, and resection should be deferred until Nature has had a chance either to fight off the infection or to establish barriers to the spread of the infection. As soon as the fluid has become distinctly purulent no delay should be allowed because of the danger of toxemia or septicemia.

TREATMENT. The general plan of treatment in these cases was (1) to first relieve the embarrassment to circulation and respiration by aspiration several hours before operation; (2) resection of the rib as near the diaphragm as possible, so as to get dependent drainage; (3) irrigation, both at the time of operation and while there was a very abundant drainage; (4) to prevent secondary infection as much as possible; (5) application of the *x*-ray in order to dry up the infection.

In the 30 cases reported ether was administered, only enough was given to permit resection of the rib and incision of the pleura; then the administration of the anesthetic was stopped and a careful exploration with one finger was made to determine the extent of the cavity.

One of the 30 cases died during the second stage of the ether from circulatory embarrassment, due to a left-sided empyema. This case had previously been aspirated twenty-four hours before operation, but upon opening the chest after death quite a large amount of pus was removed. In this case a temporary drain should have been inserted under local anesthesia, and then, after the patient's condition had improved, a resection could have been done. The sudden removal of a large amount of pus from the chest often results in great harm, for there is produced a disturbance in respiration and circulation for which heart and lungs are not able to compensate. Violent paroxysms of coughing and irregularity and weakness of the pulse are produced when there is too rapid removal, especially in cases of massive effusion. It is in this class of cases that preliminary aspiration should be done in order to give the heart and lungs a chance to adjust themselves to the change caused by the evacuation of a large amount of pus at one time. It has been shown that if the patient is placed on his abdomen during the resection there is not so liable to be set up a paroxysm of coughing and the sound lung is given a much better opportunity to functionate and the patient will take the anesthetic better.

The question as to where the resection should be done has been discussed a great deal. It has been argued that because the last portion of the lung to expand on inspiration is the portion adjacent to the sixth or seventh ribs the resection should be done at this site. Theoretically this would seem feasible, but when one realizes that the lung is covered with an exudate which is holding it more or less in a state of collapse, one can readily see that the resection should be done in the site where dependent drainage can be best established.

Of course, in those cases where the pus is loculated the drainage should be instituted as near the bottom of the abscess cavity as possible.

In the 30 cases here reported resection was done when possible in the posterior axillary line at about the level of the eighth or ninth ribs and a large fenestrated rubber tube inserted. In 2 instances, where a small abscess was found, only a cigarrette drain was inserted and it was not necessary to introduce a tube. The question as to how long the tube should be kept in can only be determined by the amount of drainage. The tube should be made shorter and smaller from time to time, and as soon as possible it should be removed, since it acts as a foreign body setting up a local inflammation.

In practically every case the pleural cavity was irrigated with either salt solution or Dakin's solution. As soon as the rib was resected and the pleura opened the patient was placed in such a position as to allow as much of the fluid to run out as possible, and then the fibrinous plugs, which were sometimes found to be twelve inches long, were pulled out with the finger and the cavity irrigated several times. The mechanical washing out of the pus and fibrinous plugs was found to be of great value.

For several days following the operation, and while there was a free flow of pus, the patients were irrigated. In one very interesting case, upon opening the pleural cavity a large amount of pus was removed. The finger was then introduced into the cavity and just opposite the opening in the chest an abscess, which had just ruptured, was found. After a few days the pleural cavity was irrigated with Dakin's solution. Immediately a violent paroxysm of coughing was set up, the patient complained of the odor and taste of chlorin and extreme cyanosis was produced, which lasted for twenty-four hours. This cyanosis may have been due to the hemolysis of the blood from chlorine poisoning, but there may also have been some interference with the respiratory circulation. In such a case, when there is a communication between the lung and pleural cavity, irrigation should not be done.

The question as to whether irrigation should be carried out has been discussed a great deal. In the cases following the more recent influenza epidemic it has been observed that irrigation was not practised and the results seem just as good. At the time of this writing, however, all the cases have not been discharged from the hospital.

There can be no doubt that the mechanical cleansing of the pleural cavity and removal of the fibrinous masses at the time of operation hastens the time of recovery.

Too much stress cannot be laid on the importance of preventing secondary infection. A great many cases of empyema continue to drain a long time because of secondary infection, which probably, in some instances, sets up a more active inflammation than the original one.

As soon as possible the patients were discharged from the wards and referred to Dr. Brown for observation and treated in the surgical dispensary. From the dispensary they were also referred to the *x*-ray department, where they were treated by application of the *x*-ray three times a week. Dr. Brown found that the discharge seemed to clear up more rapidly in these cases where *x*-ray treatment was given.

Clinical Course. The clinical course depends largely on the establishment of free drainage and the general condition of the patient. The longest stay in the hospital was sixteen weeks, the average number of weeks for the cases which recovered was six weeks and three days; but not all of them were pronounced cured when discharged from the wards. None of the 30 cases developed chronic empyema.[1] In several cases it was necessary to explore several times through the original incision; in 1 case three such explorations were made and each time a walled-off abscess was found and drained. The average time required for the temperature to reach normal was a little more than two weeks.

Summary. The etiology is perhaps the same in all cases, namely, a causative organism, which was found to be the pneumococcus in most instances and rupture of a small subpleural abscess into the pleural cavity.

The diagnosis is made by (1) physical signs and symptoms; (2) aspiration; (3) fluoroscopic and *x*-ray examination.

In the treatment the great essential factor is dependent drainage after the fluid has been found to be purulent. With this free drainage, irrigation to mechanically cleanse the cavity, exercise to aid in expansion of the lung, thus obliterating the pus cavity, the exercise of care to prevent secondary infection, and in some cases the use of *x*-ray treatment to hasten absorption of the exudate are essential factors in the treatment of these empyemas.

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[1] There were 5 deaths, 4 of which died on day of admission; 1 of these died from anesthesia during operation, 3 died without operation, on the day of admission and 1 died one week following the operation from toxemia.

CASE OF PRIMARY ENDOTHELIOMA OF THE PLEURA

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It is to be regretted that with the report of this case an autopsy record is absent, due to the refusal of the relatives to give consent. However, since the diagnosis seems sufficiently correct, as evidenced by laboratory and physical findings, and because of the rarity of the condition, it was deemed of worth in the face of this omission to report the case.

As to the occurrence of endothelioma of the pleura the following facts will impress one with its infrequency.

Clarkson reports that in 10,000 autopsies at the Pathologic Museum, Munich, there were only 2 cases of this condition found, and records show that the same number of cases were present in 2000 autopsies at the Massachusetts General Hospital. Lord, in Osler's *System of Medicine,* writes of some 40 to 50 cases sufficiently well recorded to permit of acceptance. In the Protestant Episcopal Hospital of Philadelphia there is only 1 other case on record which occurred on the service of Dr. A. A. Stevens.[1]

Men are more frequently affected than women. As to the age incidence, it is more common in the fourth and fifth decades, yet Ewing mentions a case occurring in a child of five years.

[1] Med. and Surg. Repts. of the Episcopal Hospital, Phila., 1913, i, 333.

In rare instances it has followed injury to the chest wall. Endothelioma is the most frequent primary tumor of serous membranes.

The following is a history of our case:

Joseph L., aged forty-six years; nativity: Hungary; white.

Admitted to the Episcopal Hospital, Dr. Fussell's service, July 2, 1919.

Family History. Negative.

Personal History. Measles in childhood.

Present Illness. Except for present condition the patient has always been well. He did not have influenza. This illness began about Christmas, 1918. After suffering a week he sent for his family physician. His chief complaint at that time was pain in the left chest, constant in character and aggravated by deep breathing. Failing to get relief he was sent to a hospital April 28, 1919, where he remained five weeks. While there his chest was tapped four times with a Luer syringe and a few drops of blood-stained fluid obtained each time. Their final diagnosis was acute pleurisy.

He said at no time has he ever had subjective signs of fever but has experienced some slight chilliness in the beginning of this illness; has not been able to work since the onset, although he has never been bedridden. Beginning April 1, 1919, he started with slight cough productive of mucopurulent sputum. He sleeps poorly because of pain, which he claims is worse at night. The pain is sharp, lancinating in character, beginning in the region of the left apex and radiating around the side to the left scapula. He has lost weight, although his appetite is good. He has no vomiting or headache. Bowels normal.

July 2, 1919. *Examination on Admission.* Moderately emaciated white adult of good bony framework. His appearance indicates that he has suffered considerably. Head, ears, nose and throat negative. Tongue slightly red, not coated. Mucous membranes pale. *Chest:* Anteroposterior diameter slightly increased; no bulging of interspaces; limited expansion. Slight depression of supra- and infraclavicular spaces. Vocal fremitus almost entirely absent on the left side, anteriorly and posteriorly from the apex down. Percussion reveals flatness over the same area. On auscultation breath sounds are distant and no rales are heard. There is a small area below the left scapula where modified tubular breathing is audible. The right side of the chest reveals no abnormalities. *Heart:* No shocks; normal impulse; no thrills.

Percussion revealed no departures from normal. Auscultation normal. *Abdomen:* Negative. *Extremities:* Negative.

Progress. Patient's chest was tapped on receiving ward and a few centimeters of serosanguineous fluid was obtained.

July 5, 1919. Shows crepitant rales anteriorly and posteriorly over the left apex on expiratory cough.

July 7. Pain in the left chest continues.

July 8. Pain worse last night, especially over the anterior region of left chest.

July 10. Aspiration, no fluid obtained; point of tap at the angle of scapula.

July 14. Tentative diagnosis of thickened pleura and pulmonary tuberculosis.

July 15. Pain in the left chest better; complains of cough being too dry.

July 16. Feels better; cough looser; tender over the last two ribs in midaxillary line; flinches on pressure.

July 18. Over the seventh rib anteriorly a small tender movable body was discovered and removed by Dr. Klutz, resident physician. It was about the size of a pea, tendinous in nature and buried in the muscles.

July 19. Severe chest pain all night.

July 20. A small nodule removed from the seventh interspace anteriorly, which was of the same character as one previously mentioned, and in addition was adherent to the muscle.

July 21. Another nodule removed and sent, as were the others, to the laboratory for microscopic study. No other bodies discovered.

July 23. Prefers to sleep in a chair because of orthopnea. Some abdominal tenderness; pain in the chest unchanged.

July 24. Slept well in bed; better this morning; tapped in sixth interspace, midaxillary line, and twenty-four ounces of coffee-ground fluid removed. Following this had an attack of pain in the region of the heart, which lasted two minutes and radiated down the left arm. Spat up a little blood today.

July 25. Chest *x*-rayed today; feels better and seems brighter.

July 27. Cough worse; pain in the left lower chest and abdomen on coughing.

July 29. No improvement.

July 31. Pulse rapid and weak; had a profuse sweat last night.

August 1. Feels better; continues to sweat. Camphoric acid dose doubled. Chest tapped but no fluid obtained.

August 3. Both feet edematous; complains of pain in left chest as worse.

August 4. Preponderance of edema in left leg.

August 16. Patient about as usual this morning; died suddenly at 9.15 A.M., after rounds had been made.

X-ray report (Dr. Wm. A. Newell): *Fluoroscopic examination* revealed shadow on left side of chest, dense in character from apex down, fixation of diaphragm. Diagnosis: fluid or thickened pleura. *Plates* showed heart somewhat displaced to the right; entire left chest dense; if not thickened pleura probably a growth.

Laboratory records (Dr. C. Y. White): Urine: amber; acid; 1.025; no albumin; no sugar; few white blood cells; no red blood cells; few epithelial cells; no casts.

Report of *pleural effusion:* rich in blood cells, no tubercle bacilli found.

Blood: Blood Wassermann: Negative. Blood count: Red blood cells, 2,890,000; white blood cells, 9000; hemoglobin, 72; polymorphonuclears, 66 per cent.; lymphocytes 22 per cent.; eosinophiles, 12 per cent.

Sputum always negative for tubercle bacilli.

Nodule removed from thoracic muscle showed metastatic endothelioma.

As will be noticed the first symptom complained of by the patient was the same as that occurring in ordinary acute pleurisy, namely, pain on taking a deep breath. Four months after the onset he was sent to a hospital, where he remained five weeks, and on being discharged a final diagnosis of acute pleurisy was made. The reason for this pleurisy-like pain can be understood when one considers the pathology at this stage. The process according to Ewing begins with small nodules and flat elevations widely distributed over one or both membranes. These nodules later fuse, the layers of the pleura become thickened and adherent and converted into a diffuse opaque mass covering and compressing the lung. However, the parietal pleura may not be affected. Serofibrinous or bloody fluid usually accompanies the growth. The fluid may gather in small cysts or large collections. Invasion of the lung may be entirely missing, or more often the tumor involves this organ, producing irregular nodules and masses.

This man on admission appeared to be suffering with chronic pulmonary tuberculosis. However there was a decided veil of doubt thrown over the case. The cough, loss of weight, signs of thickened pleura, for in the beginning no fluid or the smallest amounts were obtained on paracentesis, with occasional rales at apices, suggested Koch-Weeks' infection.

However a temperature usually normal and not at all characteristic of tuberculosis, negative sputum examinations and the subsequent withdrawal from a cachectic individual of a fluid dark brown in color, rich in red blood cells, together with the Roentgenologist's opinion that if the condition was not thickened pleura, it was a growth of new formation caused us to look for another explanation of the condition present. The laboratory diagnosis confirmed the suspicion of malignancy and other findings pointed to the pleura as the most likely site of onset.

In looking into the literature of this subject some interesting points were revealed; among them the fact that both pleuræ are rarely affected and that in those cases in which the condition is a diffuse involvement rather than a nodular one the appearance of the diseased membrane may be mistaken for a productive fibroid change, especially if no microscopic sections are made. This may be one reason why so few cases are found at autopsy in that some conditions called thickened pleura may be malignant neoplastic growths.

Metastases occur in the supraclavicular and axillary glands. In our case we found no glandular enlargement. The thoracic muscles are frequent sites of secondary involvement, as has been pointed out in this patient. In 6 cases on record no metastases were found at autopsy. The reason for the varying amounts of fluid obtained on tapping may be explained when one considers, as previously stated, that the fluid gathers in small cysts or large collections.

It may be of interest to state at this point that when seeking for fluid in the chest more success has been experienced on this service by inserting the exploratory needle through one of the interspaces along the midaxillary line: as in this case, after several unsuccessful attempts, a large quantity of blood-tinged fluid was evacuated when the tap was made in the midaxillary line, sixth interspace.

In summarizing, some characteristics common to most of the cases recorded are as follows:

That the process begins very much like an acute pleurisy: Loss of weight, weakness, dyspnea, absence of fever or slight increase of temperature; hemorrhagic character of the effusion, at first small in amount, later extreme; rapid reaccumulation; relief on tapping is slight or absent, and if present lasts only a short time; negative bacteriology; cachexia.

SAC OF INDIRECT INGUINAL HERNIA WITH COMPLETE OBLITERATION AT ONE POINT[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

Joseph R., aged twenty-six years, wore a truss for about eighteen months, when a small boy, for right inguinal hernia. Since childhood the hernia had not been down until it appeared as an incomplete indirect right inguinal hernia, after a lifting strain a few days before operation, which was done January 2, 1920, at the Episcopal Hospital. The sac extended below the level of the external ring and its fundus was distinct from the tunica vaginalis testis. When the sac was opened near its fundus, it was found obliterated about 4 cm. distal to the internal ring. The proximal portion of the sac, continuous with the peritoneal cavity, was then opened, and both portions of the bilocular sac excised; and the inguinal canal was repaired in the usual way (after incidental appendectomy through a McBurney incision).

Everyone knows how frequent it is to meet with partial occlusions of such hernial sacs, at one or more levels; and the occurrence of hydrocele of the cord proves that complete obliteration may occur. But the question is, does wearing a truss for eighteen months or even for eighteen years produce such an obliteration? It seems very unlikely that it ever does; certainly nothing I have ever encountered in an operation for inguinal hernia indicates that it does; and even granting that this case is an instance of the occurrence, the fact remains that the obliteration occurred not *at the internal ring*, where it might prevent recurrence of the hernia, but in the course of the sac as it passed through the canal, and that the hernia did recur in the proximal portion of the sac.

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[1] Specimen presented at a meeting of the Philadelphia Academy of Surgery January 5, 1920. Reprinted from Annals of Surgery, 1920 i, 512.

A CASE OF CARCINOMA OF THE STOMACH, WITH VERY INDEFINITE SYMPTOMS

TOGETHER WITH AN ANALYSIS OF CASES OF CARCINOMA OF THE STOMACH ADMITTED TO THE EPISCOPAL HOSPITAL SINCE 1900

By EDWARD T. CROSSAN, M.D.
ASSOCIATE SURGEON TO THE HOSPITAL

C. G., aged fifty-two years; occupation, farmhand; admitted to the medical service of Drs. Stevens and Pratt at the Episcopal Hospital on March 21, 1915; discharged March 30, 1915. Diagnosis: Bronchopneumonia.

Family History. Mother died of cancer (organ unknown). Father died of gunshot injury. One sister died of tuberculosis.

Personal History. Had typhoid fever twenty-five years ago. For the last five years has had a hernia on the left side, which he says is easily reduced and for which he wears a truss. Uses tobacco to excess. Eats much cheese, bread, bologna and fried potatoes; most of the food is taken warm.

History of Present Illness. Pain in the right axilla of one week's duration, preceded by a chill and accompanied by a cough and yellowish expectoration. No headaches or hemoptysis. Appetite fair; bowels regular.

Physical Examination. Temperature 101.3°; pulse 88, blood-pressure 112 to 85. Is well nourished; no edema; cyanosis or jaundice.

Eyes: Right pupil larger than the left, and both react sluggishly to light and accommodation.

Neck: Enlargement of posterior cervical glands.

Chest: Pneumonia areas at angle of left scapula and right interscapular line (?). Heart normal.

Abdomen: Tenderness on deep pressure in epigastrium, with muscular rigidity, but no masses. Left indirect inguinal hernia, easily reducible.

Laboratory: Urine negative. White blood cells, 9040; polynuclears, 64 per cent.

March 25, 1915. Improving. Still has "soreness" in epigastrium.

March 30. Discharged (no notes made of abdominal tenderness).

The patient was readmitted to the hospital on December 10, 1915, and discharged December 30, 1915, service of Drs. Piersol and Struse. Diagnosis: Chronic endocarditis.

Chief Complaint. Dyspnea; abdominal soreness; watery stools; vertigo and slight cough.

History of Present Illness. For one week has had diarrhea, with profuse watery stools; much mucus; severe tenesmus and some blood. Two days ago, while leading a horse, he suddenly became dizzy and fell; never had an attack of vertigo before or since. Has occasional attacks of dyspnea; cough; palpitation; has had several night-sweats. Has lost much weight.

Physical Examination. Eyes: Normal.

Heart: Enlarged to left; loud mitral systolic murmur.

Lungs: Impaired resonance anteriorly and posteriorly over the right apex, with increased tactile fremitus and vocal resonance, prolonged expiration and sonorous rales.

Abdomen: Slightly distended; no masses; slight tenderness over McBurney's point.

Laboratory: Urine shows a narrow ring of albumin and a few hyaline casts. White blood cells, 8320; polymorphonuclears, 68 per cent.

December 30, 1915. Discharged (no notes about diarrhea or abdominal tenderness.)

Third admission was on January 17, 1916, to the service of Drs. Piersol and Cole. Diagnosis: Carcinoma of liver and stomach.

History of Present Illness. Since discharge (December 30, 1915) has had frontal headache, vertigo, dyspnea, pains across the chest and diarrhea. Also has been vomiting immediately after food.

Physical Examination. Same as on last admission, with the exception that an enlargement of the liver is noted, 5 cm. below the costal margin. Also the presence of the hernia is again mentioned.

Laboratory: Urine: Narrow ring of albumin; few hyaline casts. White blood cells, 9680.

Gastric Analysis: Amount, 100 c.c.; free HCl, negative; lactic acid, positive; total acids, 43; free HCl and acid salts, 40; combined acid, 3; few red blood cells and occasional white blood cells; Oppler-Boas negative.

January 26, 1916. Still vomiting. Has petechiæ over chest and abdomen.

January 27. Transferred to surgical service of Drs. Ashhurst and Bramlett.

January 27. *Notes Made on Entrance to Surgical Ward.* History of indigestion for three years was elicited. Patient is anemic. Lately has had typical retention-vomiting twice weekly; vomitus blood-stained. There is a barely palpable mass in the epigastrium.

January 28. *Operation.* Dr. Ashhurst. Ether Anesthesia: Large mass, evidently malignant, was found in body of stomach, not involving pylorus or cardia but extending from greater to lesser curvature on both anterior and posterior surfaces and almost completely obstructing the lumen. Numerous enlarged soft lymph nodes in gastrocolic omentum; fewer and smaller in gastrohepatic omentum. The stomach was not fixed but the tumor was considered inoperable on account of the extension, so far toward the fundus of the stomach and on account of the patient's weakness. No metastasis found in the liver. Gall-bladder was normal. With great difficulty enough of the posterior wall of the fundus of the stomach was exposed through the transverse mesocolon to render a gastrojejunostomy possible; the anastomotic opening was less than 3 cm. in length.

Progress. February 19. Discharged in good condition. No unfavorable symptoms.

The patient was admitted for the fourth time on September 15, 1916, to Dr. Ashhurst's service, with the diagnosis of carcinoma of stomach and irreducible hernia. Discharged September 19, 1916, with a revised diagnosis of carcinoma of the stomach, with metastasis to omentum and hernial sac.

Personal History. Since previous discharge has been in perfect health. Has not had any pain, vomiting, diarrhea, dyspnea or edema. Says he has gained forty pounds(?), and has been doing his usual work as gardener all summer—plowing, digging, etc.

History of Present Illness. Three days previous to admission was taken with severe pain in the chest, which later migrated to the right iliac region and then became general in the lower abdomen. Has had effectual bowel movements up until the day of admission. No vomiting, fever, chills or sweats.

Physical Examination. Head and Neck: Negative.

Chest: Lungs negative. Heart enlarged to left; no distinct murmurs.

Abdomen: In epigastrium to either side of xiphoid, and extending to an inch above the umbilicus, is a firm mass, descending on inspiration and apparently not connected with the liver. Lower border of the liver not palpable. Dulness in both iliac fossæ. In left inguinal region a mass extending down into the scrotum; no impulse on coughing; not reducible.

Genitalia: Swelling in left scrotum.

Laboratory: White blood cells, 9360; polymorphonuclears, 91 per cent.

Operation: September 15, 1916. Dr. Ashhurst. Local anesthesia (eucain, 1 per cent.). Oblique inguinal incision; hernial sac found and on opening it a few ounces of straw-colored fluid escaped. No intestines or omentum found in the sac. Finger inserted through the internal inguinal ring and many nodules felt in the omentum. Sac drawn up from the scrotum and a mass of nodular character one and a half inches in diameter found on its inner surface. Sac not removed. Wound closed in layers.

Progress. September 18, 1916. Patient has not had any untoward symptoms. States that during last stay in hospital he vomited a round worm.

September 22. Examination of stools for ova and parasites negative. Appetite excellent. No pain or vomiting.

September 27. No symptoms. Wassermann negative. *X*-ray after barium shows a tumor involving greater and lesser curvatures, reducing the lumen of the stomach to the size of a pencil. Twenty-four hours later the intestine shows no obstruction; colon filled.

September 30. No untoward symptoms. Discharged.

The final admission was to the medical service of Drs. Robertson and Paul on November 4, 1916; discharged December 20, 1916. Diagnosis: Carcinomatosis.

History of Present Illness. Complains of swelling about the ankles; puffiness of the eyes; dyspnea and cough. Appetite good.

No vomiting. Some diarrhea. Marked swelling of abdomen. Losing weight rapidly.

Physical Examination. Head and Neck. Negative.

Chest: Many rales throughout both chests.

Heart: First sound at apex of poor quality.

Abdomen: Scar in upper abdomen. Over umbilicus there are two small masses the size of a dollar. Free fluid in abdomen.

Progress. November 17, 1916. Fluid in both chests.

November 18. 2140 c.c. of fluid removed from chest (side not stated).

December 8. 10,970 c.c. clear fluid removed from abdomen by paracentesis. Wassermann negative.

December 18. 3600 c.c. fluid removed from right chest.

December 20. Discharged unimproved.

February, 1917. Died at home.

The above case is exceptional in that through numerous admissions one is enabled to follow the course of the disease. At the time of his first admission he seems undoubtedly to have had a bronchopneumonia. Then, however, there was a note about tenderness in the epigastrium which doubtless was ascribed to his cough, and probably was due to that. Following his discharge he seems to have been in good health for nearly nine months, when he was seized with a group of symptoms, partly intestinal and partly cardiac.

Whether or not he had some gastric symptoms at that time is not clear, but, at any rate, they were not severe enough to attract the patient's attention, nor were there any physical findings to indicate trouble with the stomach. There was one symptom which, in a man the age of this patient, should demand attention, namely, the "loss of much weight."

The third admission was ten months after the attack of bronchopneumonia. Now the case begins to clear up, though unfortunately too late. We now find he has had indigestion for three years, that he now has retention vomiting, "loss of much weight," and a suspicious mass in the epigastrium and an absence of free hydrochloric acid in the gastric contents, all of which justified the suspicion of malignancy. The diarrhea present at this and at the previous admission is difficult to understand unless it were due to some involvement of the pancreas.

The operation disclosed the fact that the malignancy was extensive, dating from long before the second admission, and perhaps was present even at his first visit. Even though the anastomotic opening was small the gastro-enterostomy effectually emptied the stomach, for the patient had no vomiting up until the day of his last discharge, covering a period of a few weeks short of one year.

What already is well known is again emphasized here, namely, that the omentum is very resistant to malignancy, but that once it falls a prey the spread of the disease becomes much more rapid and vicious. The presence of the metastasis in the hernial sac, giving rise to the presumption of an irreducible hernia, is unique though not surprising.

Since 1900, 79 cases (exclusive of 1 reported above) diagnosed as carcinoma of the stomach, have been admitted to the surgical service of this hospital. This is an astonishingly small number, particularly as it has been variously estimated that 30 per cent. of all carcinomas occur in the stomach. The male sex was prey to the cancer in 60 instances out of the 79 cases.

The average age of the male patient was 51.3 years and the female 47.5 years. About one-third of all the cases occurred between the ages of fifty and sixty years; there were 6 cases less than forty years of age and 12 between the ages of thirty and forty-five.

Family History of Carcinoma. Such a history was only encountered 7 times. In 2 cases one or other of the parents had died of carcinoma of the stomach. In 5 of the cases the history related to the parents; of the other 2, 1 was a history of cancer of the breast in a sister and the other cancer of unknown origin in a grandmother. In 6 out of these 7 cases their own tumor was inoperable. The operable case died with carcinomatosis three months after gastrectomy.

Previous Medical History. In 3 cases there had been previous operations for duodenal ulcers (at operation the site of the tumor in one of these cases was found to be at the gastro-enterostomy). In 1 case a tumor had been removed from the uterus nine years previously. Another case had had an appendectomy three years previously for symptoms similar to the ones at admission. In only 9 cases was there a history of indigestion previous to the onset of the present trouble.

There were no notes of any value on the habits of life in the cases studied.

Symptoms. The symptom encountered in practically every case was loss of weight. Pain was found in 58 of the cases; the site of the pain was most often given as in the epigastrium and accompanied by a distress which was relieved by vomiting. In comparatively few of the cases was the pain made worse by food. In 51 cases there was vomiting and in only 8 of these was the vomitus of the coffee-ground type. There was not a single case of hematemesis, though in 1 case the vomitus was streaked with blood. In 4 of the cases there was diarrhea, and 1 case had persistent salivation. The average duration of symptoms was fourteen months; however, there was a wide variance, for there were 7 cases of three years and longer duration and 13 under three months.

An attempt was made to study separately the cases which operation proved amenable to radical measures. The symptoms in these cases were much like the average given above, with the exception that the average duration was ten months and the average age forty-nine years.

It was surprising to find that though in the majority of the cases the symptoms were of long duration, in only 9 had there been any treatment for the ailment previous to entrance in this hospital.

Physical Examination. In 49 cases a mass was found in the abdomen, practically always in the neighborhood of the epigastrium. In 11 cases there was only rigidity, in 9 tenderness in the epigastrium alone was found and the remainder were negative or the findings were not stated.

Laboratory. In all the cases but one in which the stomach contents were examined there was found an absent or greatly diminished acid content. In the exception the stomach contents were nearly normal, though at operation an extensive carcinoma was found. The Oppler-Boas bacillus and occult blood were rarely found. There was a secondary anemia in all the cases and an average of 11,900 white blood cells (27 cases). In only 9 cases was there a pathologic report on the variety of the tumor, namely, scirrhus 4, medullary 3, gelatinous carcinoma 1, cylindrical cell 1.

Diagnosis Before Operation. In 17 cases of the 51 operated on the diagnosis was not made before operation; 30 per cent. of these errors were on the side of ulcer and a like number were diagnosed pyloric obstruction.

X-Ray. *X*-ray was only made use of in 17 cases, and in only 5 of that number was the diagnosis of cancer made or verified by it.

Operation. Ten cases refused operation and 18 were considered inoperable. Of the remaining 51 operated on 14 had gastrectomies, 17 posterior gastro-enterostomies, 3 anterior gastro-enterostomies and 17 were exploratory only. In 2 cases old gastro-enterostomies were found, and, as stated before, in 1 of these a mass was found at this site; in the other the mass was on the pyloric side of the anastomosis. Metastasis to the liver was found in 11 cases and to the pancreas in 2. In 45 instances the situation of the tumor was given and was as follows:

Whole stomach	3	cases
Pylorus	23	"
Greater curvature	5	"
Lesser curvature	3	"
Anterior wall	2	"
Posterior wall	1	"
Pylorus and greater curvature	3	"
Pylorus and lesser curvature	1	"
Fundus	2	"
Cardia	1	"
Site of old gastro-enterostomy	1	"

Mortality. There were 29 deaths in this series, 22 in males and 7 in females. Seven of the 29 died without operation, 7 after exploratory, 7 after posterior gastro-enterostomy, 3 after anterior gastro-enterostomy and 5 after gastrectomy.

The mortality of the various operative procedures is as follows:

Gastrectomy	14	5 deaths, or	35.7	per cent.
Anterior gastro-enterostomy	3	3 " "	100.0	"
Posterior gastro-enterostomy	17	7 " "	41.2	"
Exploratories	17	7 " "	41.2	"

Conclusions. Manifestly this series is too small to make any deductions, but it will serve to illustrate the fact that in any person near forty, with "dyspeptic" symptoms or loss of weight, carcinoma should be seriously thought of.

In 50 per cent. of the cases here recorded no palliative or radical measure could be carried out, and in only 22.9 per cent. were radical steps possible, and that again emphasizes the point above made.

Summary. In this series of cases the family history of carcinoma, has not been very prominent. The striking fact is, perhaps, just a coincidence, that in these cases the condition was so far advanced.

There was not anything, in the previous history, of particular value.

In the series here studied, one is struck with the fact that the majority of the patients have given little thought to their "stomach trouble," for as noted before only 9 had received previous medical treatment. In every case there were symptoms referable to the stomach. (Smithies reported a series of 983 cases, 1.5 per cent. of which had no gastric symptoms.) In 64 per cent. of the cases there was found an abdominal mass on physical examination at entrance.

We find that exploratory operation has a rather high mortality here, for which no reason could be found; in this group there was an average of eight days of life following operation. At operation the pylorus alone and in conjunction with some other portion of the stomach was found involved in 60 per cent. of the cases. There was a total operative mortality of 43 per cent.

ILEOCECAL INFANTILE STENOSIS[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

Dr. Astley P. C. Ashhurst reported the following case:

Thomas B., aged two years, was admitted to the Episcopal Hospital, November 25, 1915, with symptoms of acute intestinal obstruction. The illness began November 22, with recurrent attacks of abdominal pain; during the attacks the baby gripped his belly with both fists. As he had suffered with similar attacks all during his life, no great alarm was felt. Vomiting set in, however, and on the third day of what was now seen to be the severest attack he ever had suffered he was sent to the hospital.

Examination at 1.30 P.M., soon after admission: The child lies quietly on his back, apparently exhausted. His eyes are hollow, his tongue and skin very dry. He has just vomited some bile. Since admission there have been several attacks of sudden abdominal pain, the child crying out and clutching at his belly. There is violent peristalsis. There is no rigidity, except during the attacks of pain. Above the umbilicus a transversely placed, sausage-shaped tumor is present, which is slightly movable and not very tender. A diagnosis of intussusception was made and the child was prepared for operation.

Operation. Under the anesthetic, 3 P.M. (gas-oxygen, preceded by morphin sulphate, grain $\frac{1}{48}$, and atropin sulphate, grain $\frac{1}{400}$) no such tumor as above described could be palpated. It was nevertheless determined to proceed with the operation. A right paramedian incision was made downward for three inches from the umbilicus. There was no peritonitis. The lymphatic vessels of the small and large bowels were distended. The transverse colon was normal. The small bowel was not distended. A movable mass, the size of a guinea-hen's egg, was felt in the ileocecal region. The small intestines were then packed off with pads and the ileocecal coil was delivered through the incision. No intussusception was now present, but there was a tumor in the ileum just above the ileocecal valve, about 4 cm. in length, precisely resembling the tumor seen at the pylorus in cases of infantile stenosis. There were several inflamed epiploic appendages on the cecum and ascending colon, but no enlarged mesenteric lymph nodes were detected. The wall of the ileum felt thick and the lumen appeared to be almost obliterated by the tumor, which involved the whole circumference equally (Fig. 151*a*). Apparently there had been an intussusceptson of this mass into the cecum and ascending colon. A lateral anastomosis (Fig. 151*b*) was made by suture between the ileum just proximal to the mass and the ascending colon (into the anterior longitudinal band, not as shown in the diagram into the median side of the colon). The appendix was not removed, owing to the precarious condition of the patient. The time of the operation was thirty minutes.

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[1] Read at a meeting of the Philadelphia Academy of Surgery, January 8, 1917. Reprinted from Annals of Surgery, 1917, i, 514.

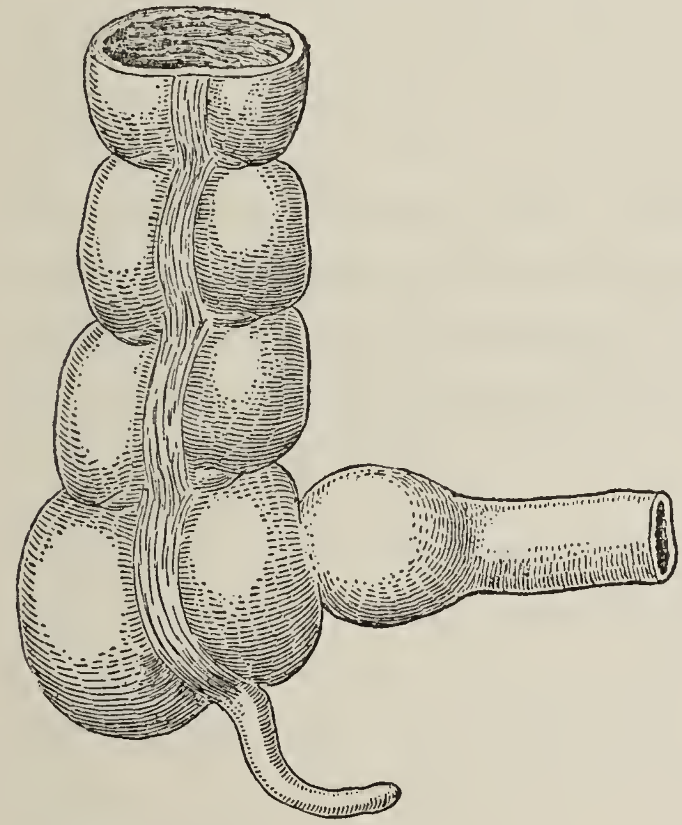


FIG. 151*a*.—Tumor of ileum proximal to ileocecal opening, causing stenosis.

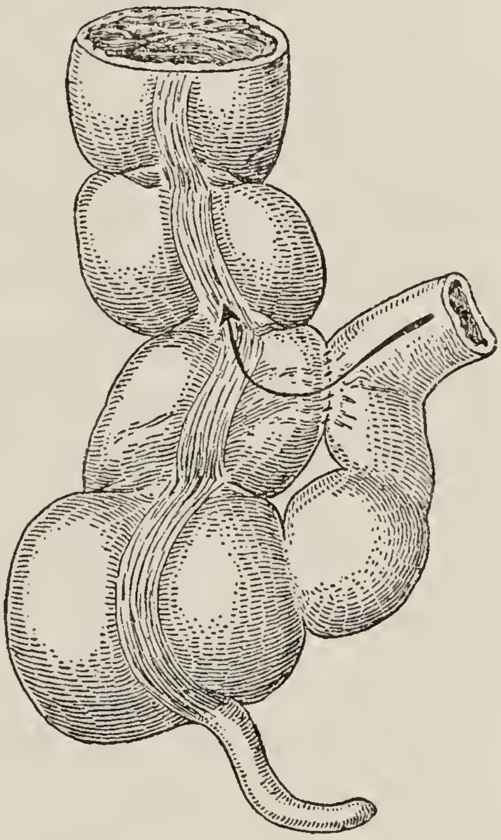


FIG. 151*b*.—Ileocolic anastomosis to short-circuit stenosis of ileocecal opening.

Recovery was entirly uneventful. The child was taken home one month later, December 25, 1915, and was seen at first at rather frequent intervals. The child is now, fourteen months after operation, in perfect health. No attacks of abdominal pain have occurred since operation.

It was not until after the operation, with its rather disconcerting findings, that it was learned from the parents that the child had been subject since earliest infancy to precisely similar attacks, none of which, however, had been so severe or had lasted so long as that which finally compelled them to seek hospital treatment.

The question of most interest is whether the tumor caused the intussusception or whether the intussusception caused the tumor. The former supposition is more reasonable in view of the fact that repeated but milder attacks had occurred more or less constantly throughout life and that the tumor had not the least evidence of being due to edema or swelling. It was as like the tumors seen in infantile pyloric stenosis as one pea is like another.

MALPOSITIONS OF THE COLON, WITH REPORT OF AN UNUSUAL CASE

By ADRIAN W. VOEGELIN, M.D.
RESIDENT PHYSICIAN IN THE HOSPITAL

In order to formulate an opinion as to whether a given position of an organ is abnormal or not, it is required that one have a definite picture of its normal location in mind. In the case of the greater portion of the large intestine this is difficult. Comparatively few individuals have a colon which will accommodate itself to the various text-book descriptions of its positions. Since these differ among themselves, considerable latitude in its course seems to be permissible before we can pronounce a colon misplaced, especially since both position and shape may vary in the same patient at different times. However, there seems to be an average type to which Nature tried to adhere.

As generally accepted, commencing at the cecum in the right iliac fossa, the colon ascends through the right lumbar and right hypochondriac regions to reach the under surface of the liver. As transverse colon it is draped more anteriorly between the epigastric and umbilical regions from the hepatic flexure to the splenic flexure in the left hypochondriac region. Then as descending colon it traverses the left hypochondriac and lumbar spaces to the crest of the ilium, where it terminates in the sigmoid flexure. The loop of the sigmoid colon, as a rule, lies wholly in the pelvis, commonly crossing over to the right pelvic wall and finally terminates at the rectum in the midline opposite the second or third sacral vertebra.

Most of the authors lay little emphasis on the frequency and importance of variations in the location of the colon, but a search through the literature of the past few years shows reports of quite a few cases. A certain percentage of abnormalities has been discovered on the operating or postmortem table, but in late years roentgenography has called attention to many cases which would otherwise have remained undiscovered because of the absence of associated subjective symptoms.

To understand fully the various wanderings from type, it is necessary to briefly consider the embryological gyrations of this portion of the gut tube. Because the intestinal canal of the embryo grows in length much more rapidly than the embryonic body, the tube becomes bent and thrown into convolutions. During the fifth and sixth weeks of intra-uterine life a conspicuous flexure appears some distance below the stomach and the bowel assumes the form of a U-shaped loop, with the closed end of the U pointing toward the ventral body wall. On the distal lower limb the cecal bud begins to form and the portion below develops into large intestine with a corresponding increase in caliber. All of the colon is still to the left of the median line, but about the eighth week the U-loop begins to rotate counterclockwise, so that its lower limb, consisting chiefly of rudimentary cecum and a part of the colon, is folded upward and over to the right side and comes to lie anterior to the duodenum and superior mesenteric artery. This moves the cecum from the umbilical region to the right hypochondriac region beneath the liver (Fig. 152, *A*).

As the colon elongates the cecum gradually descends in the right abdomen toward the iliac fossa, passing the crest of the ilium about the eighth month or later. At the same time the dorsal mesentery is shifting its parietal attachments and increases in extent to correspond with these advances.

Departures from this method of development will give rise to the congenital types of anomalies as contrasted with the acquired forms, or those forms due to a congenital condition with a superimposed acquired factor. In this matter the growth of the mesentery plays an important part. Normally the ascending and descending colons lose their mesenteries by fusion of their posterior peritoneal layer with the parietal peritoneum, but a mesocolon is found on the left side in 36 per cent. of cases and on the right side in 26 per cent. (Treves). In about 1.8 per cent. of all cases the true primitive type of ascending mesocolon persists continuous with the mesentery of the small intestine (G. M. Smith).

Black believes that the length and character of the mesentery is probably the most important single factor in causing variations in the location of the colon. A long mesentery gives greater mobility and allows the loops concerned to be displaced. Inflammation may give rise to adhesions which easily retain them in an abnormal position. This latter possibility applies to any portion of the colon.

Congenital misplacements seem to be more common on the right side, no doubt due to the less close and the later anchorage of the peritoneal attachments in this region. In this connection Morley has observed that when Jackson's pericolic membrane (a probable vestige of the embryonic great omentum) persists there is usually associated a congenitally mobile ascending colon.

Of derangement of the proximal colon the condition of cecum mobile is by far the most common. This is a state of undue mobility of the lower portion of the ascending colon whereby, as a result of an abnormally long ascending mesocolon, the cecum is permitted to migrate from its proper position. This excursion can occur to a greater or less degree with corresponding liability to impairment of intestinal function and abdominal symptoms.

Eastmond recommends making a distinction between those cases in which there is merely an abnormal descensus of the cecum when the patient is erect and those in which the mesenteric relaxation is so great that if the patient lies on the left side the cecum will fall to the median line of the body or beyond. To the former class the term "cecum mobile" applies, to the latter "wandering cecum." Though this distinction is merely one of mobility it has some importance since the question of medical or surgical treatment is largely involved.

In studying cecal mobility the possible anatomic alterations accompanying it must be considered. The condition can become of pathologic importance should the mobility and relaxation of the adjacent tissues permit the cecum to move enough to produce a kink. This kink formation can occur either in the cecum, the lowermost part of the ascending colon or in the neighboring terminal portion of the ileum. The last possibility is particularly favored if the ileum is bound down by that thickened fold of peritoneum known as Lane's ileal band which attaches the lower surface of the ileum in the region of the ileocecal valve to the parietal peritoneum near the pelvic brim. This form of obstruction as a cause of chronic intestinal stasis has, however, been much overemphasized, much as "incompetency of the ileocecal valve" was at one time.

The failure or only partial completion of the rotation of the U-loop described above is responsible for many cases of atypical location of the cecum and adjacent colon. Fig. 152, *C*, may be taken as the type of complete non-rotation. The condition is of occasional importance to the surgeon. R. H. Fowler and C. H. Mayo have each reported a series of interesting cases of appendicitis in which the physical signs appeared not in the right iliac region but as locations corresponding to where rotation of the head of the colon had been arrested.

The following constitute the majority of displacements having this origin which have been encountered:

1. (*a*) The *cecum* may not have descended to complete the rotation and will then be found in the right hypochondrium. As a result of only partial descent the cecum may be found fixed at any point between the hepatic flexure and its normal position, but usually lies upon the right kidney. In a series

of 310 adult males Robinson found 8 per cent. with undescended cecum and appendix. He found non-descent in less than 4 per cent. of females. In a few instances associated with non-rotation the cecum has been discovered in the left hypochondrium with most of the colon to the left of the midline.

(*b*) The elongation of the colon following rotation may have been excessive, causing the cecum to descend past the pelvic brim and often leaving it inaccessible and perhaps so held down beneath the pelvic peritoneum as to favor constriction of its lumen or complete obstruction.

(*c*) The ascending colon may become so long as to make secondary folds and its mesentery may be long enough to be twisted (Curschmann).

2. (*a*) Coming to the *transverse colon*, we find that ptosis is the commonest variation. If congenital it may be the only sign of enteroptosis, in which case it is rarely a source of pathologic symptoms. Or it may be only a part of a general relaxation and sagging such as occurs in those visceroptotic individuals with chronic intestinal stasis, in whom the overdistention and increased weight of the transverse limb is probably the factor responsible for the descent and elongation of this segment. The colon assumes an M-shaped course and the middle point can sink to various depths as far as the pelvis.

(*b*) The transverse colon may be shorter than normal, with an absence of the angle at the hepatic or less commonly the splenic flexure, causing the assumption of an inverted V-shaped position or some modification of the same.

Of all the cases reviewed the splenic flexure and then the hepatic flexure seem to be the most constant in their relation. These two points appear to be the mainstays in the fixation of the colon, and from a mechanical standpoint it seems necessarily so in order to give the colon a peripheral location in the disposition of its length in the abdominal cavity. Record of only one case was found in which the splenic flexure was totally wanting and the left hypochondrium was devoid of colon.

3. Folds and reduplications of the *descending colon* occasionally occur chiefly in connection with non-rotation cases in which the cecum and ascending colon are to the left of the median line with the ileum entering the cecum from the right (Fig. 152, *C*). In addition the colon has usually not acquired its full length.

Quite common are instances in which the descending colon crosses over to the right side with resultant variations in the position of the pelvic colon.

4. Displacements of the *pelvic colon* or *sigmoid flexure* are common and present many variations. Its normal length varies between ten and eighteen inches, but greater elongation is not very unusual. Marked elongation, with an extensive mesosigmoid, favors axial rotation, giving rise to volvulus of the sigmoid. In about 90 per cent. of cases the sigmoid colon lies entirely within the true pelvic cavity. In the remainder it forms a loop directed upward a variable distance toward the umbilicus, a position normally found in infancy (Morris). Displacement of the entire sigmoid to the right is not rare and usually the rectum takes a corresponding deviation to the right side.

5. The congenital nature of most of the permanent displacements of the colon has been mentioned, but in passing it is to be remembered that pressure by tumors or other organs or the traction due to inflammatory adhesions involving the colon may cause moderate and sometimes marked alterations in its course.

6. Lastly, complete transposition of the large intestine has been found in a few cases, either alone or as part of a general transposition of the viscera.

The *clinical manifestations* of displacements of the colon lie almost solely in the symptoms of intestinal obstruction and stasis which they are capable of causing. Various degrees of interference with the fecal current can exist and the factors concerned may operate continuously or at varying periods. The likelihood of symptoms arising depends directly on the liability to kink formation or similar mechanical obstruction. This in turn, in the majority of cases, is due to a mobile cecum or to kinking of the colon elsewhere brought about by unnatural mobility. Besides the mechanical factor involved in this impairment of intestinal function there is usually associated some disturbance of the sympathetic nerve supply, the constant irritation resulting in lessened tonus and activity of the muscles of the intestinal walls.

Among the symptoms characterizing cecum mobile, Sailer names attacks of colicky pain, a sense of fulness in the right side of the abdomen and often soreness in this region, sometimes giving rise to a mistaken diagnosis of chronic appendicitis. However, the tenderness found over McBurney's point is usually not so sharply localized as it is in chronic appendicitis. The cecum can often be detected by palpation and its motility demonstrated. Distinct gurgling is often felt if the mass is rolled under the fingers. Abnormal percussion findings may aid in the diagnosis. In common with all the displacements discussed an *x*-ray examination is the most important element in diagnosis. Pictures taken eighteen to twenty-four hours after a barium meal, with the patient lying on his back and then upon the left side, often show quite pronounced changes in position of the cecum. Sailer believes that medical and mechanical means will alleviate most cases and that surgical intervention is rarely indicated unless the condition has been severe for some time and has led to a dilatation and atony of the cecum.

In a small minority of colonic displacements, signs of obstruction may arise quite suddenly, but in most cases we are more likely to get the symptoms of chronic constipation—a sense of fulness in the abdomen, headache, flatulence, loss of appetite and energy. The symptoms of abdominal pain and discomfort may be more or less localized and percussion and palpation may elicit signs similar to those of cecum mobile.

TREATMENT. Medical treatment is usually efficacious in cases with moderate symptoms. This consists briefly of plenty of rest, fresh air, appropriate exercises, dietetic supervision, tonics, such as iron, nux vomica and eserin, and measures to keep the bowels well emptied of all accumulations. The latter is best effected by the use of colonic irrigations and the judicious use of laxatives. A permanent anatomic defect producing a partial obstruction to the evacuation of the colonic contents usually calls for surgical treatment, as does marked severity and chronicity of the functional disturbances.

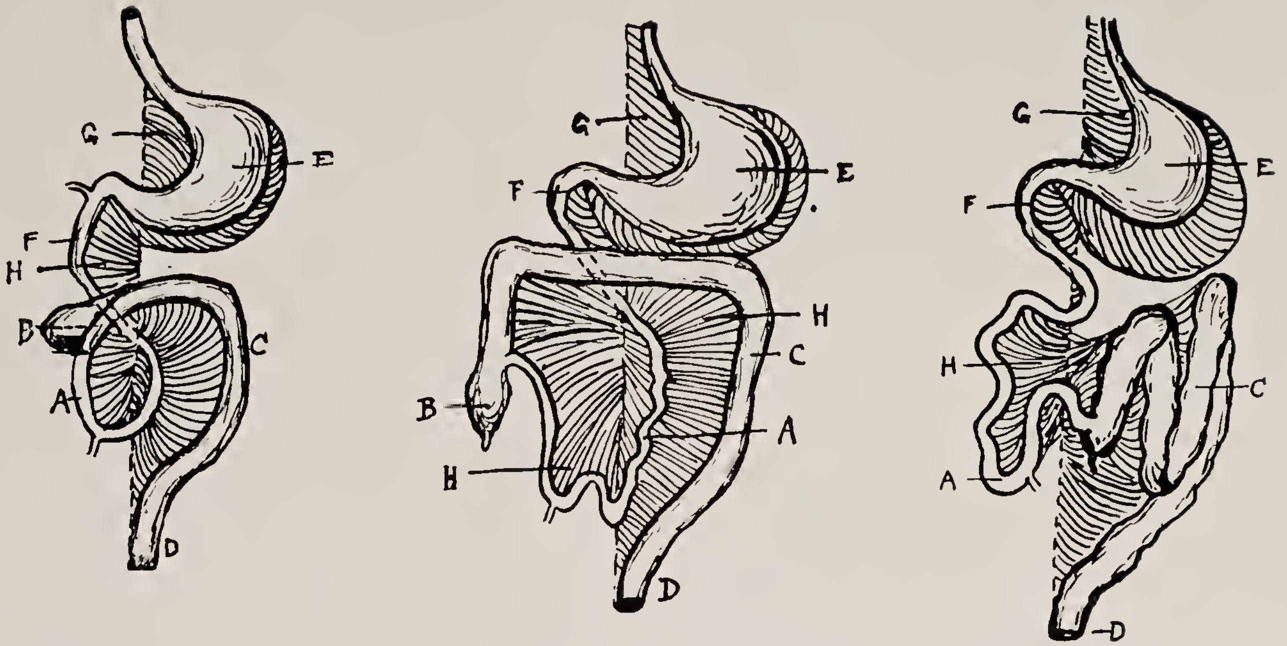
For cecum mobile the operation of cecopexy is often practised. This comprises suspension of the cecum to the parietal peritoneum as near to its normal position as possible. Peritoneal fixation of other parts of the colon can similarly be done if the displacement is due to a stretched-out ascending or descending mesocolon.

Anastomoses which sidetrack sections of the colon in an effort to redirect the fecal current in colonic stasis or ptosis are regarded as futile by many surgeons. But in cases with partial obstruction, anastomosis to provide a freer passage seems to be a rational procedure should correction of the obstruction by fixation of the bowel in its proper position be impractical or a possible source of danger. There are no statistics available to determine the comparative value of the various types of anastomoses possible. In each particular case, the course to be pursued must be decided upon from the clinical and roentgenological findings and by the condition of affairs as revealed by abdominal section when operation has been thought justified.

Illustrative of surgical treatment, I wish to present the history of the following case, which was admitted to the Episcopal Hospital on November 4, 1919:

Mr. R. M., aged fifty-eight years; nativity, United States. Occupation, laborer.

Chief Complaint. Abdominal pain and discomfort over a period of twenty years.



A *B* *C*

FIGS. 152 *A*, *B* and *C*.—*A* and *B*, normal rotation and development of the colon. *C*, failure of normal rotation. *A*, small intestine; *B*, cecum; *C*, large intestine; *D*, rectum; *E*, stomach; *F*, duodenum; *G*, mesogastrium; *H*, mesentery. *C*, failure of rotation. (After Hertwig.)

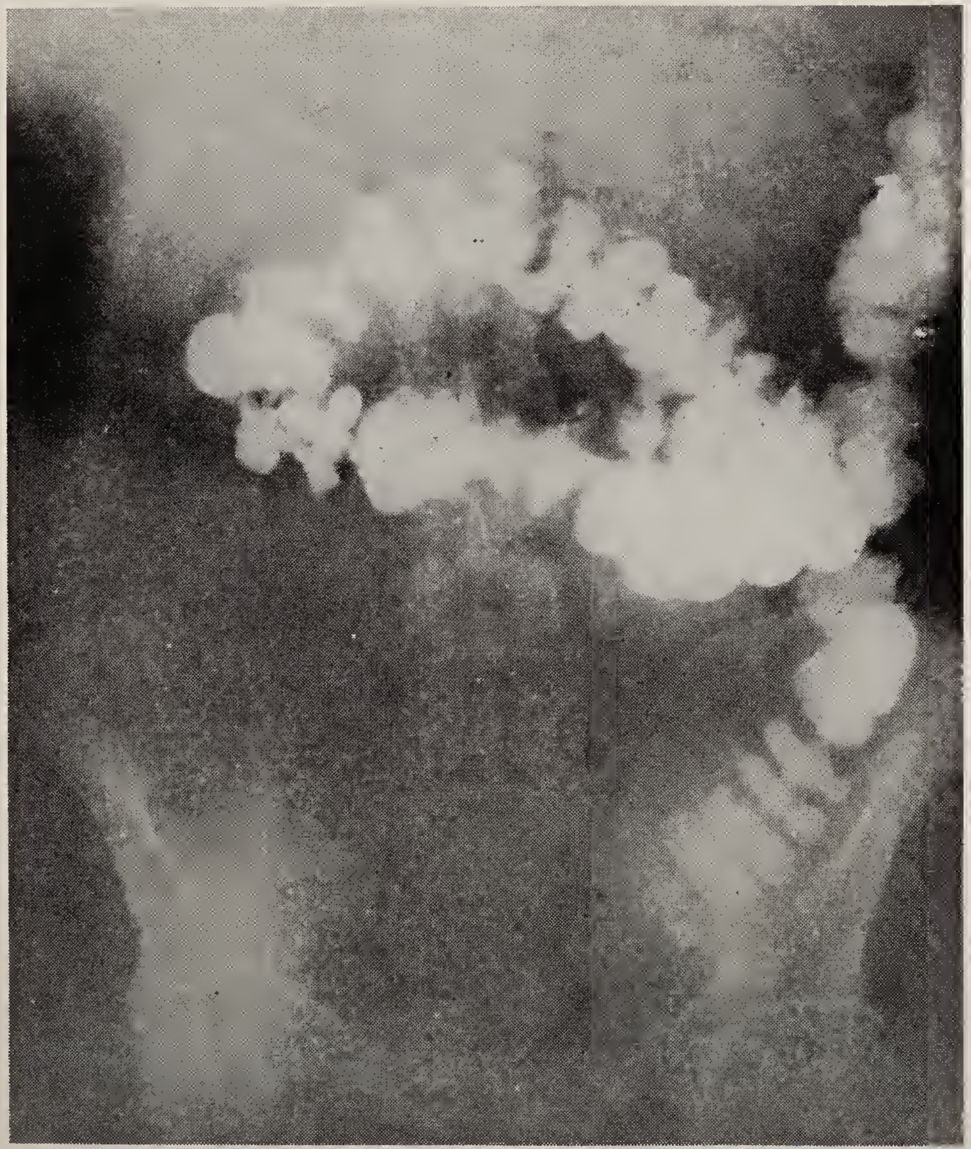


FIG. 153*a*.—Large bowel twenty-four hours after barium meal.

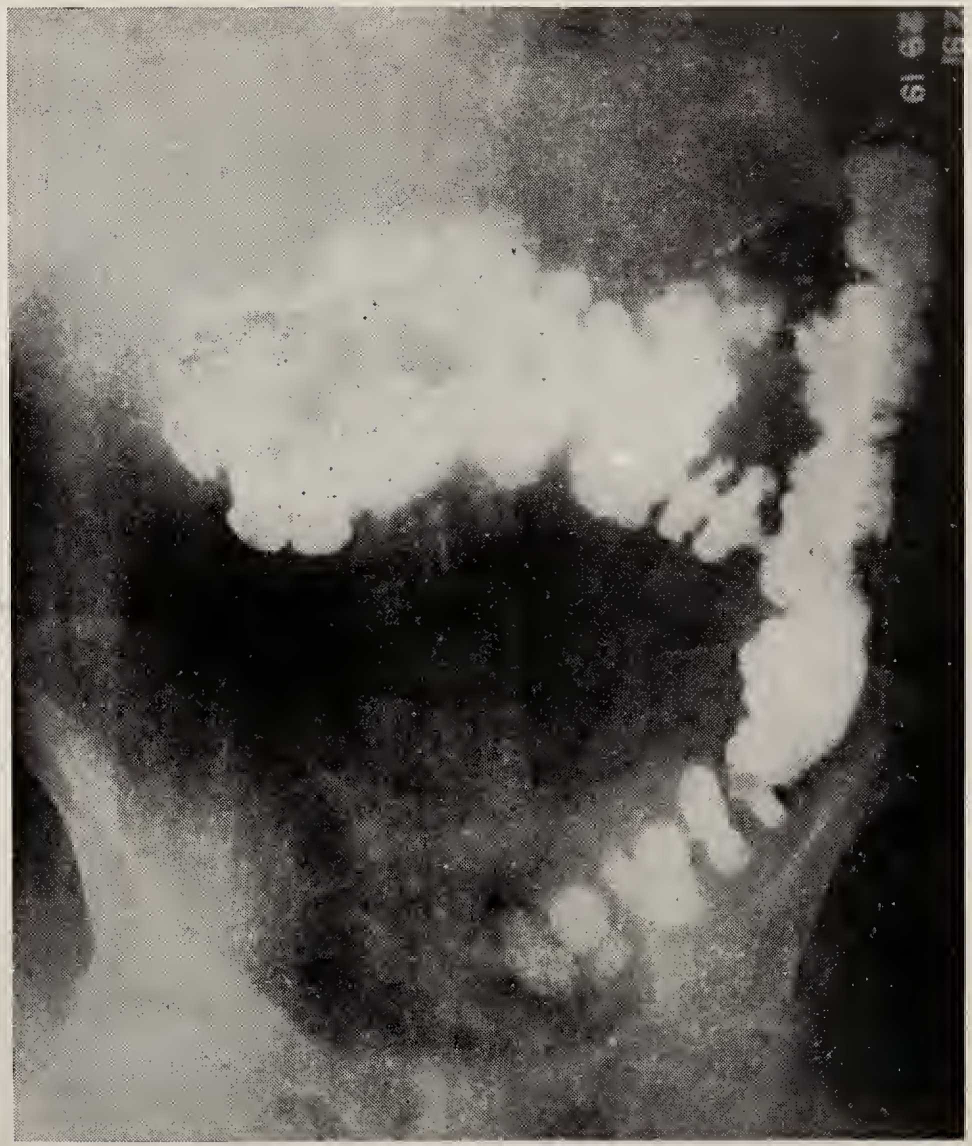


FIG. 154*a*.—Another view of large bowel twenty-four hours after barium meal.

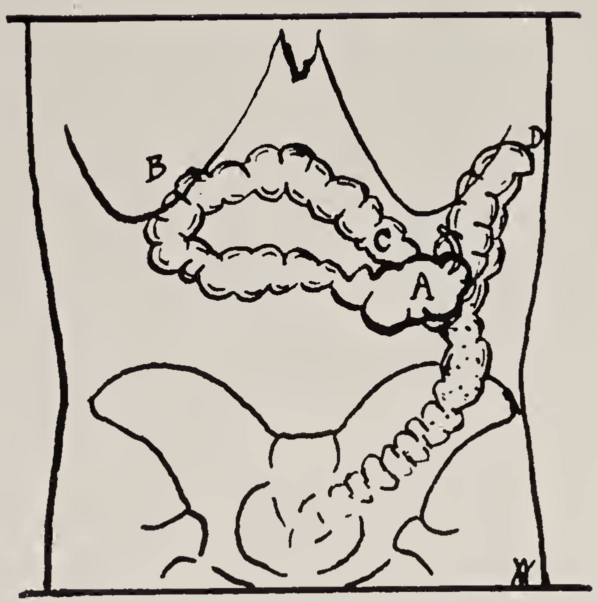


FIG. 153*b*.—*A*, cecum.

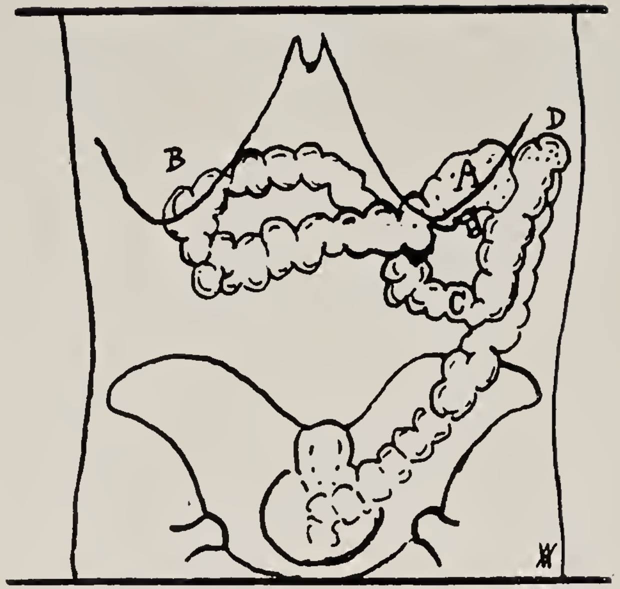


FIG. 154*b*.—*B*, hepatic flexure.

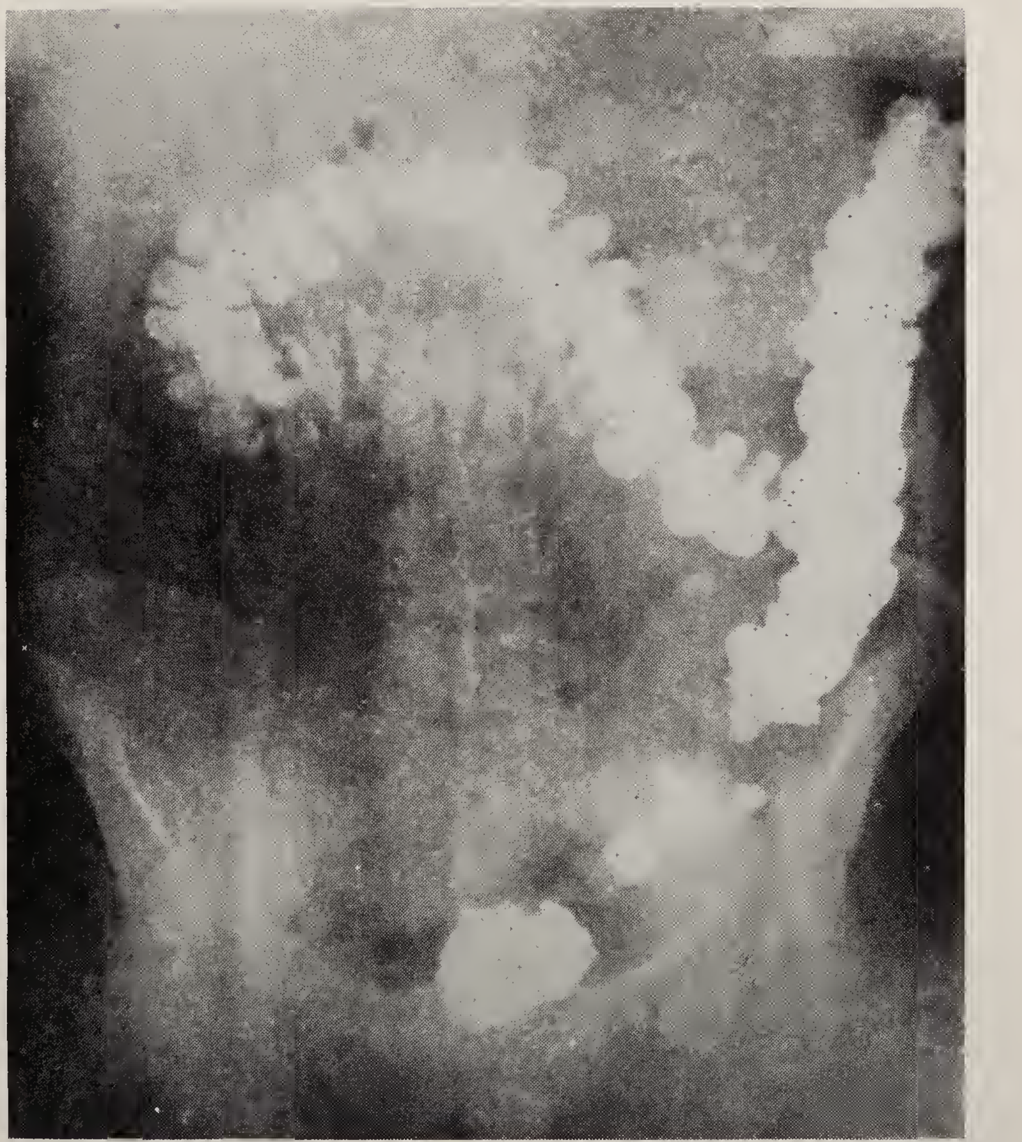


Fig. 155*a*.—Forty-eight hours after barium meal.

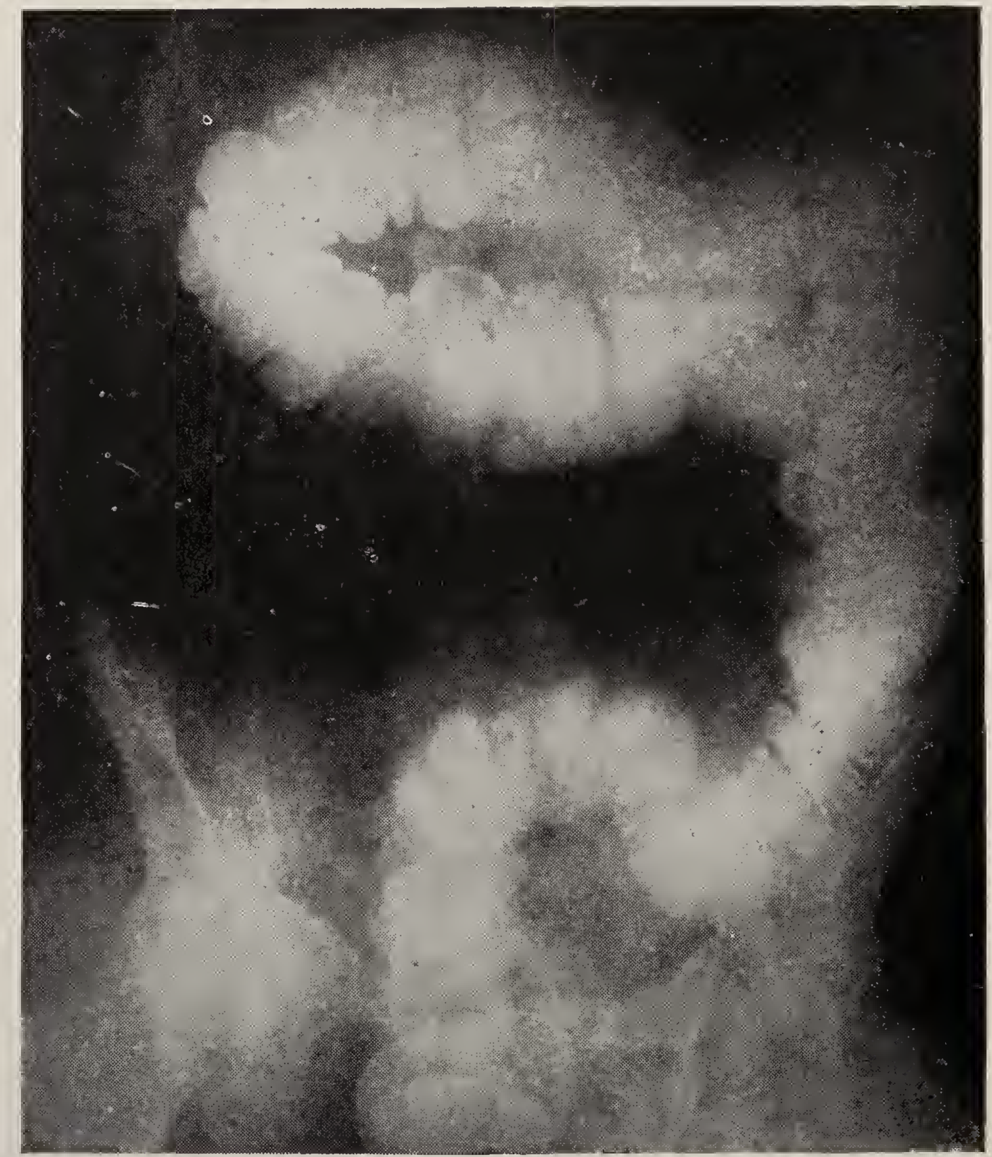


Fig. 156*a*.—After injection with barium enema.

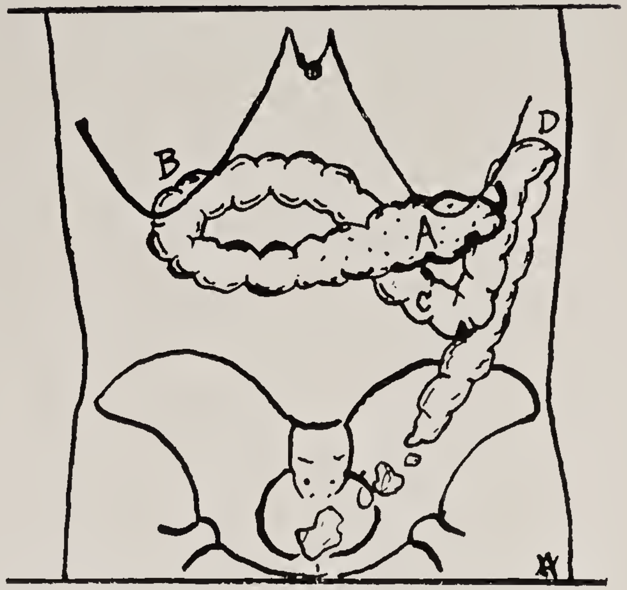


FIG. 155*b*.—*C*, transverse colon.

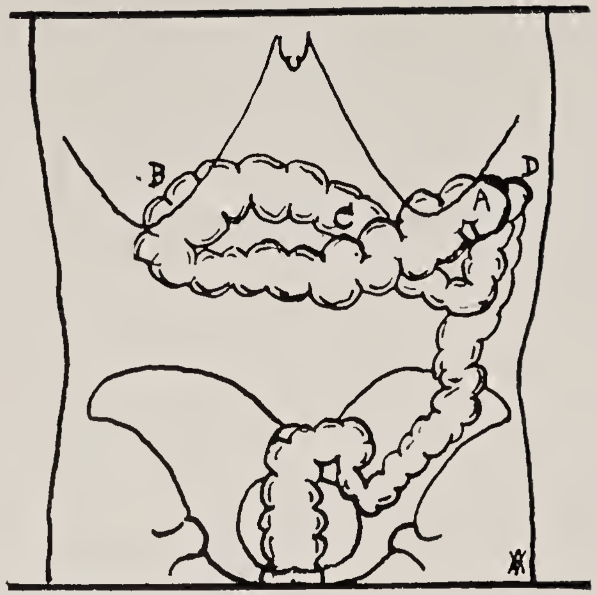


FIG. 156*b*.—*D*, splenic flexure.

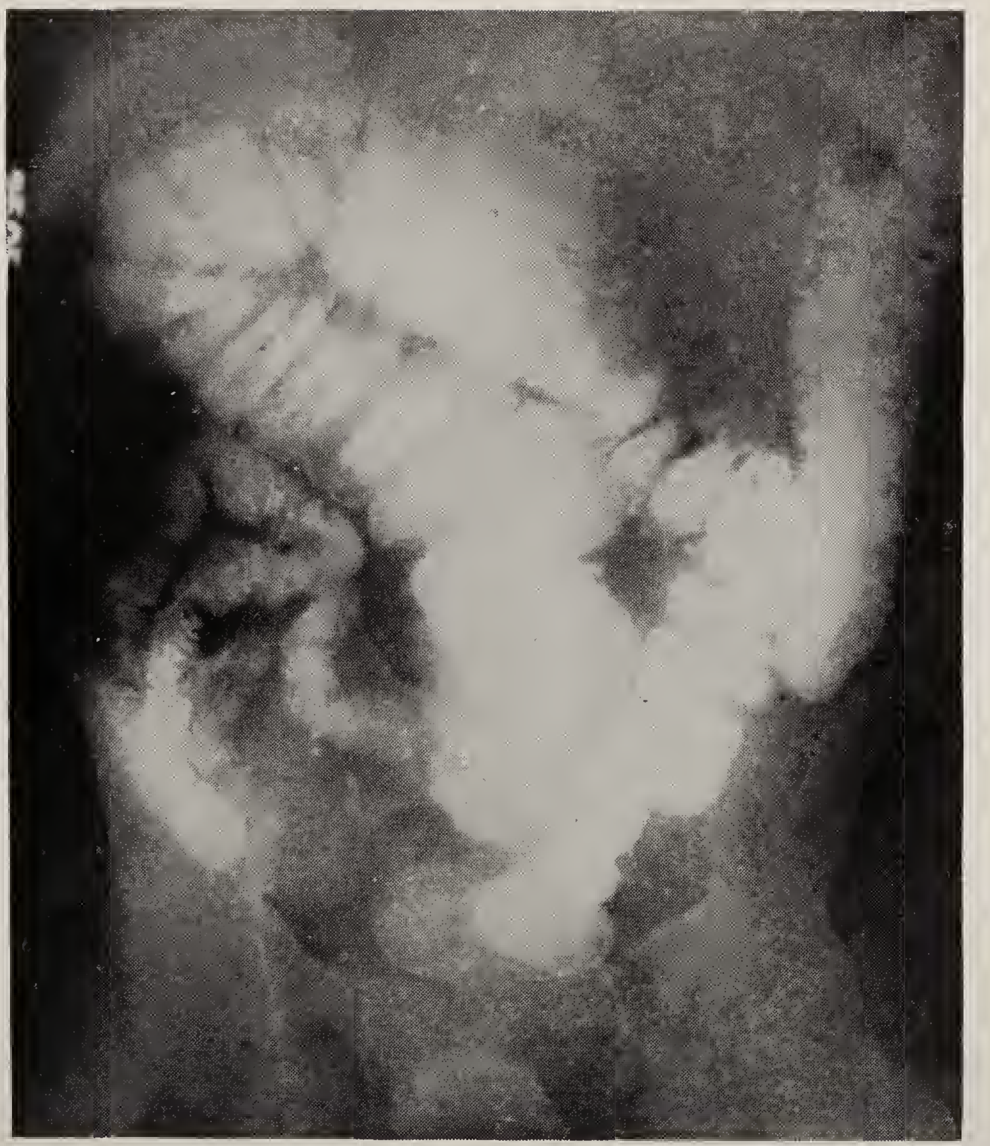


Fig. 157*a*

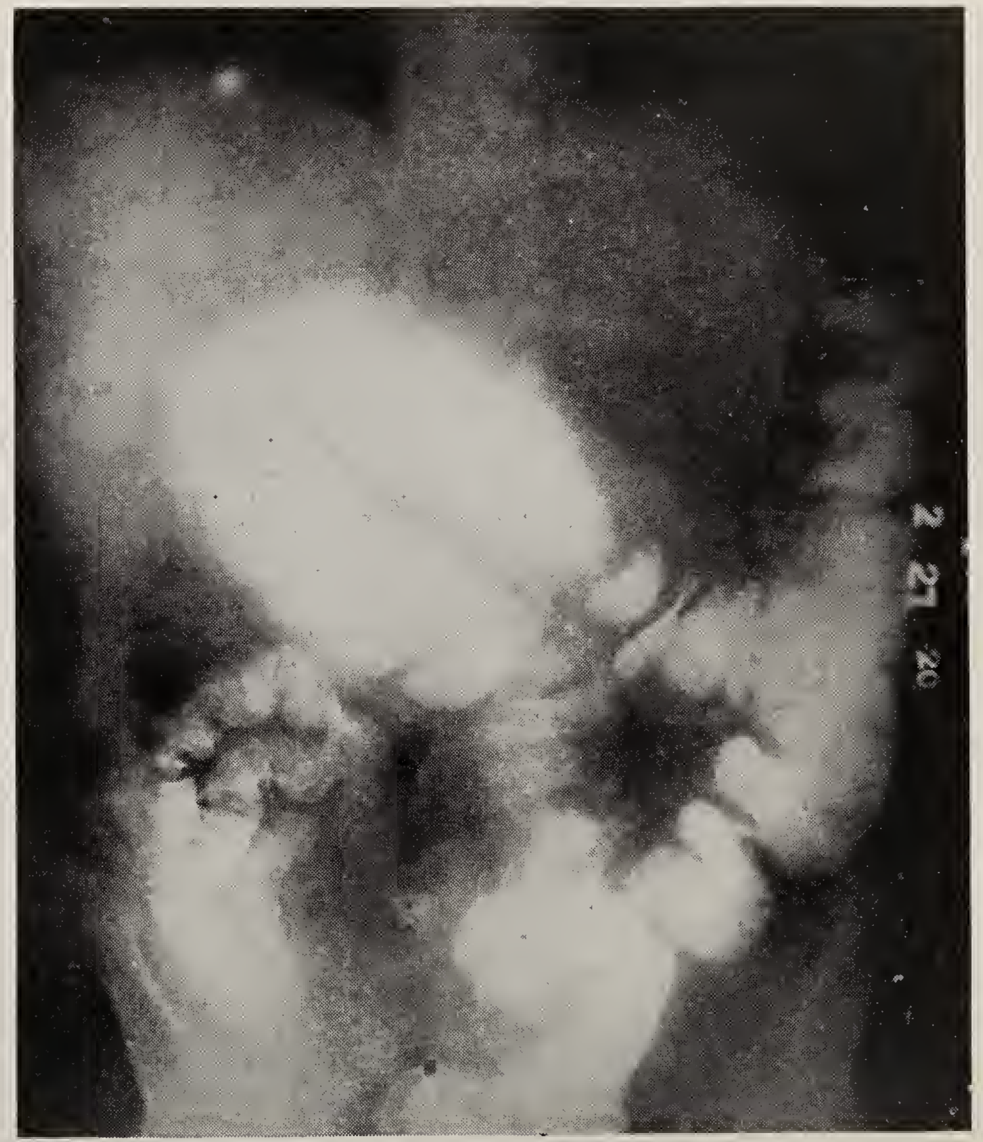


Fig. 158*a*

Figs. 157*a* and 158*a*.—Large bowel outlined by barium enema.

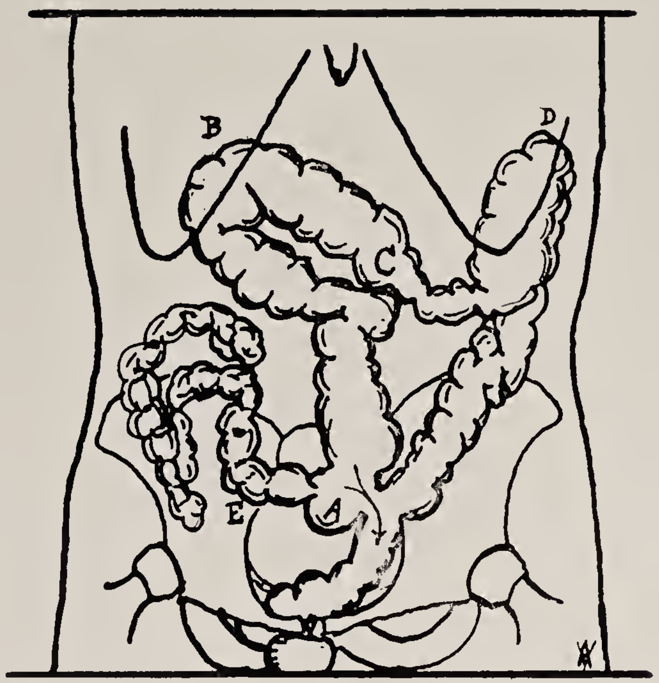


FIG. 157*b*

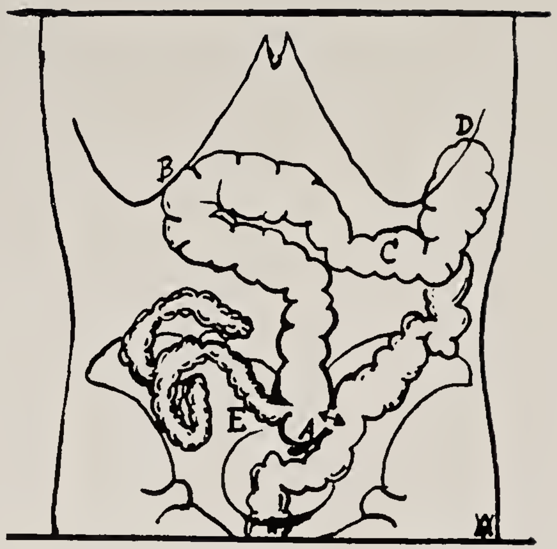


FIG. 158*b*

FIGS. 157*b* and 158*b*.—*A*, cecum; *B*, hepatic flexure; *C*, trans. colon; *D*, splenic flexure; *E*, ileum.

Family History. Wife living and well. No children. No history of malignancy or tuberculosis in family.

Past History. The patient's general health has always been good excepting for the present complaint, which has recurred intermittently for the last twenty years. He has suffered with rheumatism at intervals during the past seven years.

Present Illness. For the last twenty years the patient has suffered with recurrent attacks of pain and discomfort over the entire abdomen, but of greater severity over the left lower abdomen. Such attacks are associated with distention, gaseous eructations and discomfort around the region of the heart. The pain is of a dull, aching character, general and not localized and is apparently not influenced by the time or amount of eating. The attacks have lasted from several weeks to several months and occur ordinarily two or three times a year. After such an attack is over the patient suffers for a while with general abdominal soreness and tenderness. He also has chronic constipation and the bowels move only with purges. Appetite is good. During the last attack, which has lasted for the past six months, he has suffered from nervousness and insomnia. He states that during the last few months he has lost twenty to thirty pounds. He has never had nausea or vomiting.

On Admission. Patient is a fairly well-nourished man, about fifty-eight years old. Appears to be slightly nervous and anxious about himself.

Physical examination of head and neck negative. Thorax, heart and lungs negative. Abdomen revealed slight tenderness over the epigastrium and the two lower abdominal quadrants. No localized pain on palpation. No masses palpable. No areas of abnormal dulness. Extremities: Negative; reflexes normal.

Urine and blood examinations negative. Temperature, 98°; pulse, 96; respiration, 20.

Roentgenological Report. The first *x*-ray study was made a few days before admission. Stomach examination was negative. Pictures of the large intestine (Figs. 153*a*, 154*a*, 155*a*, and 156*a*), taken twenty-four and forty-eight hours after a barium meal, showed marked displacement of the cecum and ascending colon toward the left hypochondrium, apparently causing a chronic obstruction, which in turn causes the chronic constipation.

Operation. A cecosigmoidostomy was done by Dr. H. C. Deaver on November 6, 1919. A six-inch right rectus paramedian incision was made. The stomach and gall-bladder were found to be normal. The cecum and ascending colon were then packed off and found to be abnormally situated and freely movable in the abdominal cavity. There was complete peritoneal investiture of the ascending colon, but the site of attachment of the mesocolon was not determined. The cecum was much dilated and with its appendix was found high up in the left hypochondriac region. The appendix was rather long and partly obliterated, and was taken out in the usual manner. The cecum and ascending colon could be brought over to their natural location, but were not sutured to the parietal peritoneum, as it was probably feared the extensive fixation would not be permanently effective or curative. The sigmoidal portion of the large intestine was then brought up and a lateral anastomosis performed between its first portion and the cecum. The incision was closed in the usual manner and without drainage. No buried sutures were inserted. Deep and superficial tissues were closed with interrupted silkworm-gut sutures and dressings applied. Convalescence was uneventful except for moderate postoperative distention. Patient was discharged in good condition on December 10, 1919.

The condition of the patient as shown by the *x*-rays and by operation was one of wandering cecum. Figs. 153*b* to 156*b* represent the course of the intestine diagrammatically, and on comparing them we find there is present in each of the views a well-marked hepatic and splenic flexure (*B* and *D*). These are constant in location. Hence, taken in conjunction with the fact that the transverse colon (*C*) is found crossing the upper abdomen and anterior to the duodenum and superior mesenteric artery, we have proof that rotation of the U-loop has taken place. On picturing the cecum and ascending colon in their normal location we see that the course of the colon is then practically normal except for the rather sharp angles consistently present in the distal part of the transverse and the middle of the descending colon. Therefore, this condition can be called one of unusually pronounced wandering cecum, in which the primitive type of ascending mesocolon persisted and fusion with the parietal peritoneum failed to occur, permitting the cecum to reach its abnormal location.

The patient received a second *x*-ray examination about four months after the operation. The condition shown in Figs. 157*a* and 158*a* was found, a barium enema being used to demonstrate the anastomosis. This was injected with the patient on the fluoroscopic table. The barium was seen to pass from the sigmoid through the cecal anastomosis and rise in the ascending colon. The enema did not advance as far as the descending colon before the increased pressure caused filling of the terminal ileum and filling of the descending colon from below upward. This probably shows that there had been some obstruction in the midcolon or undisturbed ascending colon to cause the abdominal pain and discomfort other than that caused by gaseous distention.

The patient stated that he felt much better than he formerly did and had gained in weight. He had had no recurrence of the abdominal discomfort or pain since discharge from the hospital. Some degree of constipation still persisted, requiring the use of a cathartic once or twice weekly, but the patient was entirely satisfied with the result of the operation.

In concluding, I wish to thank Dr. Harry C. Deaver for permission to report this case and Dr. R. S. Bromer for the privilege of reproducing the *x*-ray negatives and for his suggestions in the preparation of this article.

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INTUSSUSCEPTION OCCURRING IN TUBERCULOUS PERITONITIS, WITH A REPORT OF TWO CASES

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A REVIEW of the literature on intussusception indicates that the condition is not infrequently caused by or associated with an inflammatory condition of the bowel, appendix or peritoneum.

In the cases which I have the pleasure of reporting there were several factors entering into the etiology of the intussusception, since along with the inflammatory process other conditions were present which probably also played a part in the etiology of the intussusception.

CASE I.—John C., male, aged twenty-seven years; admitted to Dr. Ashhurst's service at 9 P.M. on November 29, 1919. Diagnosis on admission was acute appendicitis. Duration of symptoms, twelve hours.

Family History. Negative for tuberculosis on either side of family.

Personal History. Measles in childhood. Otherwise had always been a robust man until the spring of 1919, at which time the patient began to complain of slight general weakness, loss of weight and coughing. In June, 1919, patient had a pulmonary hemorrhage and came to the medical dispensary of this hospital for an examination, and the diagnosis of pulmonary tuberculosis was made at that time. Since then the patient has been under treatment at the tubercular sanitarium at Mont Alto, Pa.

History of Present Illness. Chief complaint: Excruciating pain in the abdomen, vomiting, melena. On November 28, 1919, patient came home from the tubercular sanitarium on a visit. On the journey down he ate two whole apple pies. At 10 A.M. November 29, 1919, he was taken with sudden acute abdominal pain, vomiting at the onset, no chills or fever, but with passage of blood by the bowel. The pain remained all day, being knife-like in character and located to the right and just above the umbilicus. The family physician, Dr. William R. Brown, of the surgical dispensary of this Hospital, was called in to see the patient at 6 P.M. and referred the case to the hospital with a diagnosis of acute appendicitis.

Condition on Admission. Fairly well nourished adult male, aged twenty-seven years. Patient suffering extreme agony, with legs flexed on the abdomen, vomiting. Temperature, pulse and respirations normal.

Head: Slightly coated tongue. Eyes show rather an anxious expression.

Neck: Negative.

Chest: Heart action regular, with sounds of good quality. No enlargement; no murmurs.

Lungs: Expansion generally limited on both sides. Phthisical chest. Resonance greatly impaired in the upper two-thirds of the right lung; bronchial breathing and fine crepitant rales. Left apex also shows some infiltration.

Abdomen: Slight distention of the lower abdomen. Marked rigidity of both recti muscles more particularly on the right side. General abdominal tenderness, worse on the right side and slightly above the umbilicus.

Extremities: Negative.

Patient was sent to the operating room and operated on immediately.

Operation (by Dr. Hawfield). Appendectomy; resection of the bowel: Suture of intestinal perforation. Anesthesia: gas-oxygen; duration, three and a half hours. (Dr. M. C. Bond.)

Right rectus incision, 10 cm. long, finger introduced and the appendix was found to be acutely inflamed and slightly adherent. No free pus in the abdomen. Small fecal concretion was felt. The appendix was removed, inverting the stump. The finger was then introduced into the pelvis and a small, rather resistant mass in the bowel was palpated and the bowel was found adherent. The incision was prolonged and some of the coils of intestines were delivered and found to be studded with numerous small tubercles, and were somewhat distended. About 30 cm. from the lower end of the ileum a small perforation of a tuberculous ulcer was found and was repaired with linen. Numerous tuberculous ulcers were found along the lower portion of the ileum; upon delivering more of the bowel the mass previously palpated was found about three feet from the ileocecal junction. The portion of the bowel above the mass was adherent and delivery was not attempted. The portion below the mass was collapsed. The mass was firm and very resistant, and resembled somewhat an enterolith, but was recognized as an intussusception. No neck of the intussusception could be made out, due to the numerous adhesions. The intestines were carefully packed away, except that portion of the bowel involved in the intussusception, and an opening was made longitudinally, about 4 cm. long, down on the apex of the intussusceptum. The apex was found to be gangrenous, bleeding and sloughing and a resection of the portion of the bowel involved was decided upon. The sloughing portion of the bowel was tied off and cut away, in order to keep the field of operation as clean as possible. The bowel was then resected, including the entire portion of the intussusception, and an end-to-end anastomosis with the Murphy button was made. The small hole in the mesentery thus made was sutured together in order to prevent hernia of the bowel through this hole. A glass drainage tube was introduced into the pelvis and the incision closed in layers.

The patient lived for ten days after the operation. During that time he had almost continuous hiccoughs, except when he was under the influence of morphin. Gastric lavage was given daily but this only relieved the condition temporarily. The abdomen was opened through the incision after death and a leakage was found around the anastomosis, the patient having died of general peritonitis.

Case II.—Mary B., female, aged three years, was admitted October 16, 1910, to Dr. Frazier's service, being referred by Dr. F. P. Bigley. Diagnosis on admission: intussusception. Duration, about thirty-six hours.

Family History. Negative for tuberculosis.

Personal History. Always a small, delicate child. For the past three months patient has been having attacks of distention of the bowel and constipation.

History of Present Illness. The day before admission to the hospital the patient was seized with acute pain in the abdomen, vomiting and distention of the bowel. Her condition was somewhat relieved by an enema, but on the evening of admission the pains became more severe and there was ballooning of the abdomen and constant vomiting. The temperature remained normal and the leukocyte count 7000.

Condition on Admission. Poorly nourished child. General appearance that of tuberculous peritonitis. A mass was felt over the cecum. A great deal of abdominal distention present. Diagnosis of intussusception was made and an operation decided upon.

Operation (by Dr. Ashhurst). Reduction of intussusception. Release of adherent Meckel's diverticulum. Suture of two intestinal perforations.

Anesthesia: Ether. Duration: Forty-five minutes.

Paramedian incision to the left of the umbilicus extending almost down to the pubis. Clear serum on opening the abdomen. One coil of the intestines prolapsed through the wound and was found to be covered with numerous miliary tubercles. This coil was replaced and the intestines were packed away by gauze. All the parietal peritoneum, small intestines, cecum and colon were covered with tubercles. The omentum formed a hard, tough, oblong mass resembling the pancreas, gathered up into a bunch, in front of the transverse colon. A mass was found at the cecum, around which were many adhesions. The mass was delivered and the intussusception was reduced. The intussusception involved the ileocecal coil, the ileum having entered the cecum for 2.5 or 3 cm. This was easily reduced, being unadherent. The appendix was normal and was not removed. As the terminal ileum above the intussusception was collapsed; it was evident that there was an obstruction higher in the small intestine. This was found about a meter higher in the ileum, and above the obstruction the small intestine was distended. In the lower collapsed portion of the ileum were many strictures from tuberculous ulcers and numerous friable adhesions. The obstruction was caused by a red band-like Meckel's diverticulum, adherent across the jejunum from below upward and binding the coils of intestine against the spine. The tip of the adherent Meckel's diverticulum was freed from the spine, while its base remained attached to a neighboring coil of the bowel (ileum). All the coils were adherent, and on releasing the diverticulum there was an escape of gas and feces from among the friable intestines. Two perforations in the intestines were found (perhaps a spontaneously developed entero-anastomosis) and were sutured with difficulty. The bowel was rinsed with hot saline, and an iodoform gauze drain was introduced. The abdominal wound was closed by through-and-through sutures of silkworm gut.

Patient died three days later of peritonitis. Exploration of wound showed a fibrinous exudate throughout the abdomen; no abscess; no obstruction. The sites of suture of the bowel were not recognized.

In the report of the above two cases it will be seen that in both instances there were found to be numerous adhesions, and tuberculosis of the peritoneum along with the intussusception. Whether the intussusception was a cause or an effect of the fatal peritonitis in each instance it is difficult to determine. A complicating peritonitis from a tuberculous perforation of the intestine into adhesions, which was present in both cases, might well be a predisposing factor in the development of an intussusception. On the other hand, the numerous strictures of the bowel noted in the second case, might in themselves be sufficient cause for the development of an intussusception, as the result of disordered peristalsis. With the inflammatory process, which is diffuse in tuberculous peritonitis, there are, in most instances, either recent or old adhesions of the bowel to themselves or to the adjacent organs; and the mechanical interference to peristalsis and to the passage of the contents of the bowel may easily give rise to conditions favorable to the production of an invagination. In most cases of tuberculous peritonitis, there is an associated tuberculous enteritis, and sometimes, as in the first case which I have reported, also a tuberculous appendicitis. The ulceration of the bowel not only weakens its coats, but also has a disturbing influence in the peristalsis of different areas of the bowel, causing an irregular peristalsis, and thus may lead to the production of an intussusception. Indiscretions in diet, as in the first case reported, will aid in causing irregular and excessive peristalsis. The fact, however, that intussusception is such an unusual complication of tuberculosis of the peritoneum indicates that ulceration of the bowel alone is not a sufficient cause, unless other factors are present also. It is possible that in cases of abdominal tuberculosis where there are apt to be enlarged lymph nodes present in the mesentery an invagination may be produced by a large lymph node pressing in the coat of the bowel, as a tumor would, thus giving rise to a small invagination, which may go on to a definite intussusception. A great many cases may not survive after tuberculous infection long enough for the development of abdominal lesions sufficient to cause an obstruction, or the obstruction may be too slight to cause any definite symptoms, other than constipation. This was the condition in the second case reported for the three months preceding the development of the acute obstruction.

Intussusception occurring in appendicitis is very infrequent, and in those cases in which it occurs, the vermiform appendix probably acts as a polyp or by traction on the bowel.

ACUTE ABDOMINAL SURGICAL COMPLICATIONS OF TYPHOID FEVER[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

At the Episcopal Hospital, Philadelphia, there have been done 86 laparotomies on patients with typhoid fever since the first operation for typhoid perforation, July 1, 1898. Of the whole number of patients 15 recovered and 71 died, a general mortality of 82.5 per cent.

In 3 cases no lesion was found: 2 of these patients recovered without further unfavorable symptoms and 1 died, the course of the disease being uninfluenced by the intervention.

In 3 cases peritonitis was present, but the causative lesion was not found at operation. All these patients died: in 1 a perforation of the ileum was found at autopsy; in 1 a perforation of the gall-bladder was found at autopsy; and in 1 no autopsy was made.

In 4 cases peritonitis was present without actual perforation of the intestinal tract; 3 patients died and 1 recovered. In the first fatal case the abdomen was full of foul-smelling, bloody fluid and there were two large gangrenous areas involving the entire circumference of the lower ileum; these were packed off with gauze, but death followed in three hours. In the second fatal case there was a large gangrenous intussusception, which was resected, death occurring twelve hours later; autopsy showed the sutures firm and the anastomosis (end-to-end) patulous, but another intussusception had formed higher in the small intestine. In the third fatal case a suppurating mesenteric lymph node had ruptured. In the only one of these four patients who recovered there was excess of abdominal fluid, the intestines were highly injected and there were several thin ulcers, but no perforation. The abdomen was closed without drainage after free irrigation and the patient's convalescence appeared to have been accelerated by the operation.

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[1] Verbal communication to the American Surgical Association, June, 1915. Reprinted from the Transactions, vol. xxxiii, p. 165.

In 5 cases the appendix alone was affected: 4 times by an incidental appendicitis (appendicectomy in all), with 1 recovery and 3 deaths; and once by what appeared clinically to be a perforation of a typhoid ulcer in the appendix, although histologic examination showed evidences of diffuse appendicitis. This latter patient recovered after removal of the appendix. In addition to these 5 cases of isolated disease of the appendix, the appendix was perforated in one case and highly inflamed in another, in each of which there was also a perforation of the ileum. The appendix was removed in both cases and the perforation of the ileum was sutured. The patient with the inflamed appendix recovered, but the case of perforation of the appendix terminated fatally.

In 3 cases there was a lesion of the gall-bladder. In the first of these an empyema of the gall-bladder was drained, but the patient did not recover. In the second case, already mentioned, the perforation of the gall-bladder was found only at autopsy, while in the third cholecystectomy was done for perforation and the patient recovered.

In 1 case a ventrosuspension of the uterus was done for pelvic symptoms in a patient who developed symptoms of typhoid fever immediately after operation, and recovered after a typical attack of the disease.

In 1 case there was a perforation of the transverse colon, which was sutured, but the patient died.

In 68 cases there was perforation of the ileum. Eight patients recovered and 60 died, a mortality of 88.2 per cent.

Among these 68 patients there were 10 with more than one perforation of the intestinal tract. All these 10 patients died; 8 had two perforations in the ileum; 1 had three perforations in the ileum; and 1 had a perforation of the appendix. Another patient, who died, had intestinal obstruction high in the small bowel (from a kink) in addition to a perforation of the lower ileum. In one of the fatal cases already mentioned (the first ever operated on at this Hospital), the perforation was found only at autopsy. In all the other 67 cases the perforation was found and sutured.

If only the cases are considered in which a lesion (perforation of intestine, gall-bladder or appendix lesion, threatening perforations, etc.) was found and adequately treated there were 80 cases, with 12 recoveries and 68 deaths, a mortality of 85 per cent.

This mortality, which is higher than that reported from some other hospitals, is difficult to account for. If, however, the cases are grouped according to date a slight improvement is noted during the last six years:

	Total.	Recovered.	Died.	Mortality.	
1898–1903 (inclusive)	23	4	19	82.6	per cent.
1904–1908 . .	44	5	39	88.6	"
1909–1915 . .	13	3	10	77.0	"
Total . . .	80	12	68	85.0	"

During the second period tabulated above there was a severe epidemic of typhoid fever in Philadelphia, happily terminated in 1908 by the introduction of filtered water into that part of the city where the Episcopal Hospital is situated. During the years 1903 to 1907 inclusive, for example, the annual number of admissions for typhoid fever averaged about 1000 cases; since 1908, on the contrary, when water filtration entered as a factor, the number of yearly admissions for typhoid fever has averaged about 144 cases. And yet during the last period, although the number of typhoid admissions has so wonderfully decreased, the total number of yearly admissions to the Episcopal Hospital has increased about 50 per cent. Especially noteworthy is the average annual number of typhoid perforations observed:

Average Annual Number of Typhoid Perforations at the Episcopal Hospital, Philadelphia. Six years, 1898–1903, 3.83 annually; five years, 1904–1908, 8.8 annually; six and a half years, 1909 to June 1, 1915, 2 annually.

This is a shining example of the truth of the maxim that prevention is better than cure: during the last six years *there have been prevented annually nearly seven cases of typhoid perforation.*

There is one other point to which I desire to call attention. This is the advisability of attempting to prevent perforation in suitable cases by a precocious operation, the operation consisting in establishing a false anus above the seat of most marked intestinal lesions in the lower ileum. This is not a new proposal, and though I have not been able to persuade any of the physicians who were in charge of typhoid patients to let me attempt the operation, I am firmly convinced that it has its place. The following is an illustrative case:

Toward the end of August, 1912, I was called to the medical ward to see a lad, aged eighteen years, then in the end of the second week of his disease, for threatening symptoms of intestinal perforation. The boy was very toxic, shivering constantly; and there was some abdominal pain and slight rigidity. But there had been no sudden change in his condition, and I had to state my candid opinion that I did not think perforation had taken place. I suggested, however, that it was a suitable case to establish a false anus in the ileum above the level of greatest disease, in the hope that drainage of the intestine might not only prevent perforation subsequently but also relieve his extremely toxic state. My advice was not accepted.

The boy's temperature continued to range from 103° to 104° F., and no improvement in his condition took place. A few days later, September 1, he had eight hemorrhages from the intestine during the day, a total amount of fifty-six ounces of blood being passed. On September 4, at 5 A.M., he vomited. He seemed worse to the resident physician; and was more rigid over the right iliac fossa. I was called again to see him at 1 P.M. He had been having opium for the hemorrhages, and was barely conscious: he was dull, speechless and helpless-looking. The abdomen was moderately distended, *very* rigid, especially on the right below the navel; there was no peristalsis. During the examination he had a sudden attack of propulsive vomiting (bile only), without nausea. I made a diagnosis of peritonitis from perforation or a leak without actual perforation.

Operation was done at 4.30 P.M. Ether anesthesia. Right rectus incision of two inches gave exit to foul-smelling, blood-stained fluid. In the ileum, close to the cecum, were two gangrenous areas, that nearer the cecum surrounding the entire circumference of the bowel for a distance of four inches. There was no actual perforation. The smaller area of gangrene was inverted into the lumen of the bowel and the larger was packed off with iodoform gauze. The time of the operation was now eight minutes. A glass tube was passed to the pelvis and left for drainage. Two pints of saline solution were given intravenously before the patient left the table. Death occurred four hours later. The culture of the peritoneal fluid gave a mixed growth, including the colon bacillus.

I believe that operation was justified a week earlier and that it might have saved the patient's life.

My own individual experience in operation for the acute abdominal complication of typhoid fever may be summarized thus:[1]

Lesion.	Cases.	Recovered.	Died.
Perforation of ileum	6	1	5[2]
Appendicitis	2	1	1[3]
Perforation of appendix	1	1	0
Perforation of gall-bladder	1	1	0
"Laparotomie blanche"	1	1	0
	11	5	6

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[1] Since June, 1915, I have operated on two more patients with typhoid perforation of the ileum, both of whom died, about one week after operation.

[2] Two patients died from second perforations, occurring after operation. Two were moribund at time of operation and died within four hours. One lived thirty hours.

[3] Case of diffuse peritonitis. Died after eight days from typhoid toxemia, not from peritonitis.

REPORT OF A CASE OF SPONTANEOUS EVACUATION OF A GAUZE SPONGE FROM THE PERITONEAL CAVITY BY WAY OF THE BOWEL[1]

By JOHN P. JONES, M.D.
FORMERLY RESIDENT PHYSICIAN IN THE HOSPITAL

WE have all seen or heard of cases in which the surgeon has been unfortunate enough to leave a sponge in the abdominal cavity. Usually its presence has promptly made itself known by a variety of disquieting symptoms which have led the surgeon to open the abdomen for the second time for its removal, much to the surgeon's embarrassment and the patient's discomfort. Although such an occurrence is unfortunately not infrequent, I believe it is extremely unusual that Nature should remove a sponge from an abdomen which has been tightly sutured in four layers, especially when its removal is effected without the aid of surgical interference and in such a way that the patient afterward regains perfect health.[2]

While chief of the surgical service in Evacuation Hospital No. 36, A. E. F., France, such a case came under my observation, the patient having been transferred to this hospital from another after operation. The following is the history:

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[1] Reprinted from the New York Medical Journal, 1919, cx, 941.

[2] When formerly on duty in the Surgical Dispensary of this Hospital a woman attended for some weeks after a laparotomy, with vague abdominal complaints. After a considerable absence she returned, presenting a gauze pack which she claimed had been passed by the anus; her abdominal but not her mental symptoms were relieved.—*Editor.*

Case History.—Mr. X., a well-developed and nourished white man, who had always been in good health except for the so-called diseases of childhood, was admitted to Hospital No. —, February 4, 1919, with the classical symptoms of acute appendicitis. On the following day an acutely inflamed catarrhal appendix was removed through a three-inch McBurney incision. The appendix was retrocecal and was very difficult to free from adhesions, necessitating the introduction of several gauze packs and sponges into the peritoneal cavity during the fifty-minute operation. There was also an acute perityphlitis with recent adhesions. A very small gauze drain was inserted into and behind the cecum emerging from the lower end of the incision. At the time of sewing up it was stated that the sponge count was correct.

February 6, 1919. General condition of the patient fair, considerable vomiting, and some pain in the belly.

February 7, 1919. Patient still complained of some abdominal pain. Vomited two or three times during the preceding night, had a slight amount of abdominal distention. All liquids by mouth were discontinued. Temperature and pulse record were as follows:

	Temperature		Pulse	
Date	A.M.	P.M.	A.M.	P.M.
Feb. 5, 1919 . . .	97·4°	98·0°	80	72
Feb. 6, 1919 . . .	98·8°	99·0°	88	90
Feb. 7, 1919 . . .	99·2°	99·4°	104	94
Feb. 8, 1919 . . .	99·8°	98·4°	88	92
Feb. 9, 1919 . . .	98·6°	98·8°	72	80
Feb. 10, 1919 . . .	98·8°	98·2°	96	92
Feb. 11, 1919 . . .	98·2°	98·2°	96	80
Feb. 12, 1919 . . .	98·4°	99·0°	84	80
Feb. 13, 1919 . . .	98·8°	99·0°	84	88
Feb. 14, 1919 . . .	99·1°	99·6°	100	104
Feb. 15, 1919 . . .	99·2°	100·2°	80	96
Feb. 16, 1919 . . .	98·3°	99·0°	100	110
Feb. 17, 1919 . . .	98 4°	99·2°	100	98
Feb. 18, 1919 . . .	98·6°	98·0°	92	84
Feb. 19, 1919 . . .	98·6°	98·6°	96	98

February 9, 1919. Drainage gauze removed and a light wick drain inserted through the skin only. Some nausea was felt by patient during the afternoon, but he did not vomit.

February 10, 1919. No change except patient again complained of considerable pain in the abdomen. Its nature or location was not recorded.

February 15, 1919. General condition of the patient was good; wound perfectly healed. The patient was put on light diet though he had very little appetite.

The patient made a slow recovery, complaining of occasional pains in the abdomen. His general appearance was good though he insisted that he did not feel at all well. He was urged to get out of bed, but he was slow in doing so, and was really not up and about until the fifth week after operation. After this he gradually increased the amount of his exercise but stated that he did not feel well, could not regain his strength, had occasional cramp-like pains in the abdomen, and had periods of rather obstinate constipation. As his general appearance was good and as nothing abnormal was revealed by physical examination the ward surgeon was inclined to believe the patient was simply lazy.

At times his bowels would move normally every day and at other times there would be periods of several days when it was necessary to give rather large doses of purgatives. The bowel movements were invariably preceded by severe cramp-like pains in the abdomen. The stools were usually well formed and of normal color. By the aid of purgatives his bowel movements were fairly regular until April 5. They did not move from that time until April 11, though he was given several aloin, belladonna and strychnine pills and one or two doses of castor oil.

During each of these six days he had several attacks of severe cramps in the abdomen, which would last for a few minutes and gradually subside. Their point of maximum intensity was usually the epigastrium, but often they would be diffused over the entire abdomen. The patient was so ill on April 11 that he was confined to his bed the greater part of the day. During that day he was given two large doses of castor oil, and in the early evening he passed an immense stool composed of feces and a gauze sponge.

On the following day the patient stated that he felt like a new man; his appetite was good and he passed two well formed stools without the aid of a laxative. After this his convalescence was rapid and uneventful except for the passing of two small gauze shreds about a week later. His appetite continued to be good, his bowel movements were regular and he gained strength rapidly.

Examination of Sponge. The sponge was saturated with feces. It was rolled and wadded into a comparatively small mass, and on being spread out was shown to maintain its two original longitudinal folds. Unfolded it measured 40 by 85 cm. It was not disintegrated to any great extent and it tore with almost as much resistance as a new piece of gauze. Pus cells could not be found, and it gave a negative occult blood test. The stain of feces could not be washed out of it.

I think there can be no doubt that the sponge was passed by rectum, as the patient passed the stool into a bed pan, and it was discovered at once by a very trustworthy orderly. It does not seem possible that the patient could have swallowed a sponge of this size during the anesthetic; furthermore it was not of the same size as the gauze squares furnished in this hospital for use during anesthesia. It is presumed that the sponge was left at the side of, or more probably behind, the cecum in contact with the raw surface left by the loosening of adhesions. Granulation tissue soon became incorporated into the meshes of the gauze and the combined action of pressure and a low grade infection devitalized the bowel wall, causing sloughing. The sponge thus found itself in the gut lumen, while the peritoneal cavity was protected by adhesions caused by the irritation of a foreign body.

A CASE OF RETROPERITONEAL CYST, PROBABLY ORIGINATING IN THE WOLFFIAN BODY

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

AND

JOSEPH H. McGUIRE, M.D.
FORMERLY RESIDENT PHYSICIAN IN THE HOSPITAL

Retroperitoneal cysts are of sufficient rarity to make the report of cases of some interest. They are to be distinguished from *mesenteric* and *omental cysts*, from *echinococcus cysts*, from *dermoid cysts*, and from *cysts of traumatic origin* (encapsulated hematomas), any of which may be more or less retroperitoneal in location. Most cysts arising in the parovarium or epoöphoron develop within the layers of the broad ligament, and are situated in the true pelvis. Should remnants of the Wolffian body fail to descend to their usual site, however, a cyst developing from them may be situated higher, even up near the kidney. The following paragraphs succinctly describe parovarian cysts, and the resemblances between the typical cases and the atypical case herewith reported become evident:

Parovarian Cysts.—The *parovarium* or *epoöphoron* lies in the broad ligament between the ovary and Fallopian tube. It is formed by the remains of the Wolffian body (Fig. 159), and is composed of a longitudinal tube (*Gärtner's duct*), and transverse tubules which run from the hilum of the ovary to join the longitudinal tube. The *Hydatid of Morgagni*, which is present in about 50 per cent. of females, is recognized as the lateral continuation of the longitudinal duct; it enters the broad ligament on its anterior surface between Fallopian tube and ovary. *Kobelt's tubules* are the aberrent tubules of the Wolffian body between the hydatid of Morgagni and those tubules which enter the hilum of the ovary. Any of these tubular structures may become the seat of cystic formation. Cysts arising from the hydatid of Morgagni and from Kobelt's tubules usually are small, are attached to the lateral border of the broad ligament by a more or less distinct pedicle, and seldom produce symptoms; they are to be distinguished from myxomatous and cystic degeneration of the fimbriæ of the Fallopian tube.

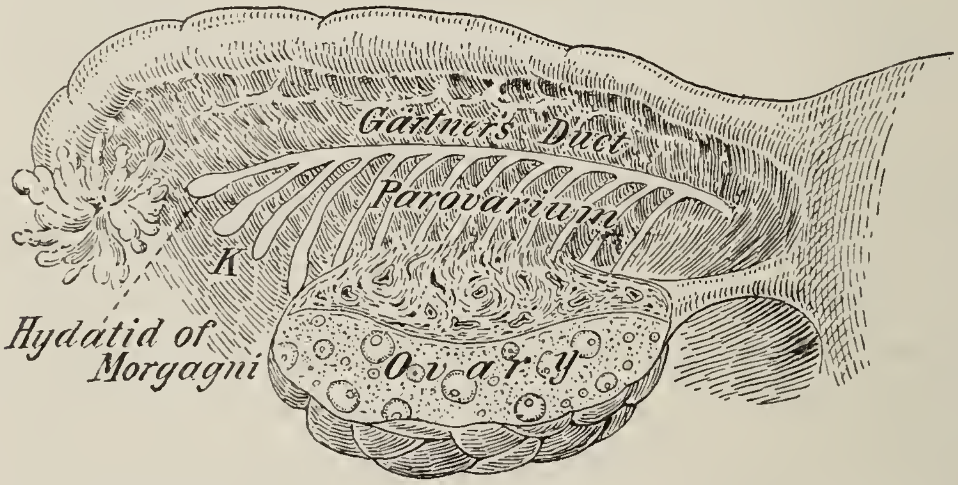

Fig. 159.—Diagram of ovary and parovarium. (Ashhurst's Surgery, courtesy of Messrs. Lea & Febiger.)

The *typical parovarian cyst* forms about 10 per cent. of all cases of ovarian cyst. It develops and spreads within the folds of the broad ligament (hence it is known as the "broad ligament cyst"), almost always is unilocular, grows slowly, and seldom attains very great size. Its contents are clear, "like spring water," and the cyst wall is lined with a single layer of cylindrical epithelium. It is easily distinguished from an ovarian cyst because it is independent of the ovary, is covered by peritoneum, possesses a double layer of vessels on its surface (one belonging to the peritoneum and the other to the cyst wall), usually is easily enucleated (rarely forming adhesions), possesses no distinct pedicle, and almost invariably has the Fallopian tube stretched out over its surface at some distance from the ovary.

A Case of Retroperitoneal Cyst

Carrie J., aged twenty-six years, was admitted to the Episcopal Hospital July 31, 1919 (Dr. Ashhurst's service).

Family History. One healthy child, living and well.

Previous History. For the past three years has had sharp pain in right side of abdomen, if she lifted a heavy weight, took a quick step up stairs or turned suddenly in bed. Pain was sharp, but not knife-like or excruciating and was only of instantaneous duration. No previous attacks of abdominal pain associated with vomiting, chills or fever. No previous abdominal pain of any duration in her life. One week previous to entrance a mucopurulent vaginal discharge developed. (Husband developed acute urethritis a few days later.)

Present Illness. Today, July 31, 1919, at 10 A.M., while doing her housework as usual, was taken with sudden severe abdominal pain, more severe in right side. Went to bed but could not move, as pain was so severe. Pain relieved by morphin given by the physician summoned. Vomited at onset. No chill and did not feel feverish. Entered hospital at 4 P.M., July 31.

Examination. Pain and tenderness in the right abdomen. Spasm of right rectus. Mass, size of a lemon (?), palpable in region of cecum. Mucopurulent vaginal discharge was profuse. Vaginal and rectal examinations not made. Ice-bag applied locally, with relief of pain.

August 1. No pain present. Slight local tenderness. Abdominal rigidity persists. Mass as on entrance. Vaginal examination shows retroversion and thickening of tubes.

Diagnosis. Chronic salpingitis and retroversion.

Operation (Drs. Ashhurst and McGuire): Ether. Under ether a mass is seen in right iliac fossa, the size of fetal head, rising to above the umbilicus. Thinking an appendicular abscess had been overlooked, on account of rigidity, a transverse incision (15 cm.) was made at level of spine of ilium. On opening peritoneum a cystic mass presents, feeling like a pregnant uterus but is softer. Uterus normal in size, retroverted not adherent. Salpingitis both sides (subacute). The mass is retroperitoneal, extending from the brim of true pelvis to below kidney. The kidney feels normal. The mass has displaced the ascending colon and cecum to right. The posterior parietal peritoneum slides over the cyst wall freely (Fig. 160*b*). Anterior parietal peritoneum is sutured to that which covers the cyst wall (Fig. 160*c*), because the acute inflammatory onset of the present illness made it possible that the cyst was suppurating.

Cyst is punctured and fluid "like spring water" gushes out. Amount of fluid over one liter. Cyst wall enucleated from the parietal peritoneum. There seemed to be no pedicle. The cyst wall was adherent at the point of eversion (Fig. 160*d*), and a ligature was applied here. Cigarette drain.

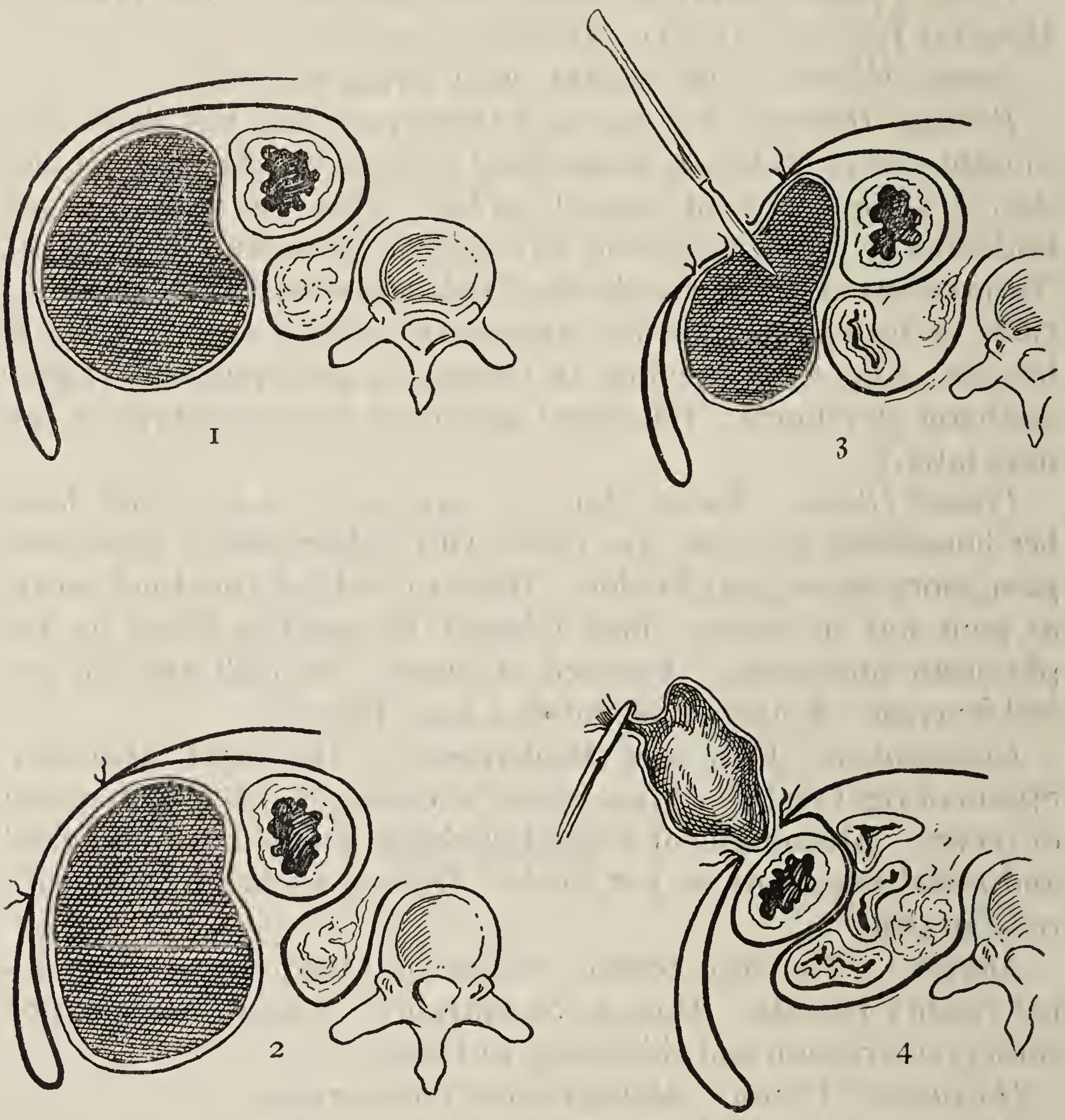

Fig. 160.—Diagrams to illustrate a case of retroperitoneal cyst.

1. Above on the left is shown in transverse section the relation of the cyst to the ascending colon, which it has pushed toward the vertebral column.

2. Below on the left, the visceral and parietal layers of peritoneum over the cyst have been sutured together.

3. Above on the right, the cyst is being opened.

4. Below on the right the cyst wall, after its evacuation, is being enucleated, allowing the ascending colon and coils of small intestine to resume their natural positions.

Pathologic Examination. Smear and culture of fluid; no organisms, no growth. Microscopic examination of cyst; cyst wall composed of fibrous tissue lined by a single layer of columnar epithelium (Dr. C. Y. White).

August 3. Drain removed.

August 10. Incision healed.

August 16. Discharged from hospital.

Six months later patient stated she had been free from abdominal pain since operation, except slight pelvic pain at time of menses. Slight mucopurulent vaginal discharge persisted between periods.

An abstract of a quite similar case recently reported by La Pointe (Bull. Soc. Chir., Paris, 1919, xlv, pp. 728 and 790) is appended:

La Pointe presented a retroperitoneal cyst, the size of the head of a newborn baby, multilobulated but unilocular. The wall was thin, almost transparent, like that of a parovarian cyst. In no point was there any solid tissue. He had removed this cyst from a woman, aged thirty-four years, who had already undergone an abdominal hysterectomy ten years previously, for adnexal disease. The cyst presented in the right flank, infringing on the iliac fossa by its lower pole and not quite reaching the false ribs by its upper pole. The mass was fluctuating, somewhat mobile, and dull to percussion. He thought at first that he had to do with a cyst developed at the expense of the right ovary, not removed at the former operation. But the true pelvis was perfectly free, and by pushing the tumor upward it became prominent in the loin. He made a tentative *diagnosis of hydronephrosis* in a very low-placed movable kidney.

Accordingly he operated by the lumbo-iliac route, as if on the kidney, and up to an advanced stage of the operation he continued to regard the tumor as a hydronephrotic kidney. He had to traverse the fatty capsule of the kidney to expose the tumor and enucleate it. This was easily accomplished, except below and in front, where the cyst was adherent to the cecum and ascending colon, and to the neighboring peritoneum, which was opened and again closed. When enucleation was complete the cyst fell out into his hands, for it had no pedicle. Then only did he perceive that it was not a case of hydronephrosis but a retroperitoneal cyst, and he saw the normal kidney at the upper part of the cavity left by removal of the cyst.

Although at this time no histologic examination of the tumor had been made, the diagnosis must, he said, rest between (1) a cyst of the mesonephros or Wolffian body and (2) a cystic lymphangioma.

Later (p. 790) he reported that histologic examination of the cyst by Lecène showed it to be lined by cubical epithelium, with slightly staining nuclei, and with protoplasm staining deeply with eosin. The appearance of the epithelium resembled closely that of the glomerules or canals of the Wolffian body, as they are seen in the epoöphoron or parovarium.

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SPERMATOCELE, WITH REPORT OF A CASE[1]

By EDWARD T. CROSSAN, M.D.
ASSOCIATE SURGEON TO THE HOSPITAL

Spermatocele is accepted today as meaning a retention cyst of the scrotum, containing spermatozoa, and arising either from the vasa efferentia, the canal of the epididymis or from the embryonic remnants around and about the testicle and lower end of the cord. Like many other medical terms it is not quite exact, for it is claimed that spermatocele can exist without spermatozoa, and furthermore there are a few cases on record in which the cyst extended beyond the external inguinal ring. In view of the varied opinions as to the genesis and etiology it would be better to define it as a cyst originating in the scrotum and which is, or has been, in communication with the semen-carrying system.

History. The term spermatocele appears to have been primarily employed by Guérin, in 1785, in reporting an obscure inflammatory condition of the testicle, which in no way bore any resemblance to what is now recognized as spermatocele. In the literature of that period these cysts were doubtless diagnosed as encysted hydrocele.

In April, 1843, Liston wrote an article the title of which was "Some Observations on Encysted Hydrocele." In this article he mentioned two cases in which the fluid withdrawn from these sacs contained spermatozoa. Two months later Lloyd reported three similar cases, the first of which was revealed when he was using some fluid from an "encysted hydrocele" to dilute blood for microscopic examination. These findings were confirmed by Paget, Curling, Sédillot, Gosselin and others, and though these proofs seemed to point toward a pathologic entity, the term encysted hydrocele survived until 1860, when Cavasse used the term spermatocele as applying to cysts containing spermatozoa.

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[1] Reprinted from Annals of Surgery, 1920, ii, 500.

The subsequent studies of this condition were conducted chiefly by the French and Germans, and to Dolbeau, Hanusa, Kocher, Luschka, Hochenegg and von Hoffman we owe much of the information which we possess.

The American literature is very scanty on the subject. Ellis, in 1858, reported the finding of spermatozoa in hydrocele fluid. From that date until 1876 we find no further reports; in the latter year Dr. William Hunt and Dr. John B. Roberts reported a case which had been diagnosed as hydrocele, but on aspiration they found the fluid was of a milky color and the microscopic examination showed spermatozoa.

Following the above records only an occasional report is found until 1907, at which time Whitney published a thorough review of the literature, and then comes a hiatus which extends to the present year.

Anatomy. To understand the origin and etiology of spermatocele, a review of the relation of the epididymis and testicle, and of the embryonic remnants of the epididymis and testicle is quite essential.

The testicle hangs suspended in a space, the cavum vaginale, from which it is separated by the visceral layer of the tunica vaginalis. The latter is continued on to the epididymis, at the lateral margins of which it is reflected forward as the parietal layer, and as this is more extensive than the visceral layer, the above-named cavity results. The testicle is enclosed in a thick capsule of fibrous tissue, the tunica albuginea. The tunica albuginea sends prolongations inward, dividing the testicle into lobules. Each lobule contains the seminiferous tubules, extending from the base where they end blindly, toward the apex, at which point they unite to form the tubuli recti. The rete testis is formed in the mediastinum testis by the tubuli recti, and from it spring twelve to fifteen vasa efferentia, which pierce the tunica albuginea and pass backward in the ligamentum epididymis superius to form the coni vasculosi in the globus major. The canal of the epididymis arises from the coni vasculosi and at the cauda becomes the duct of the vas deferens.

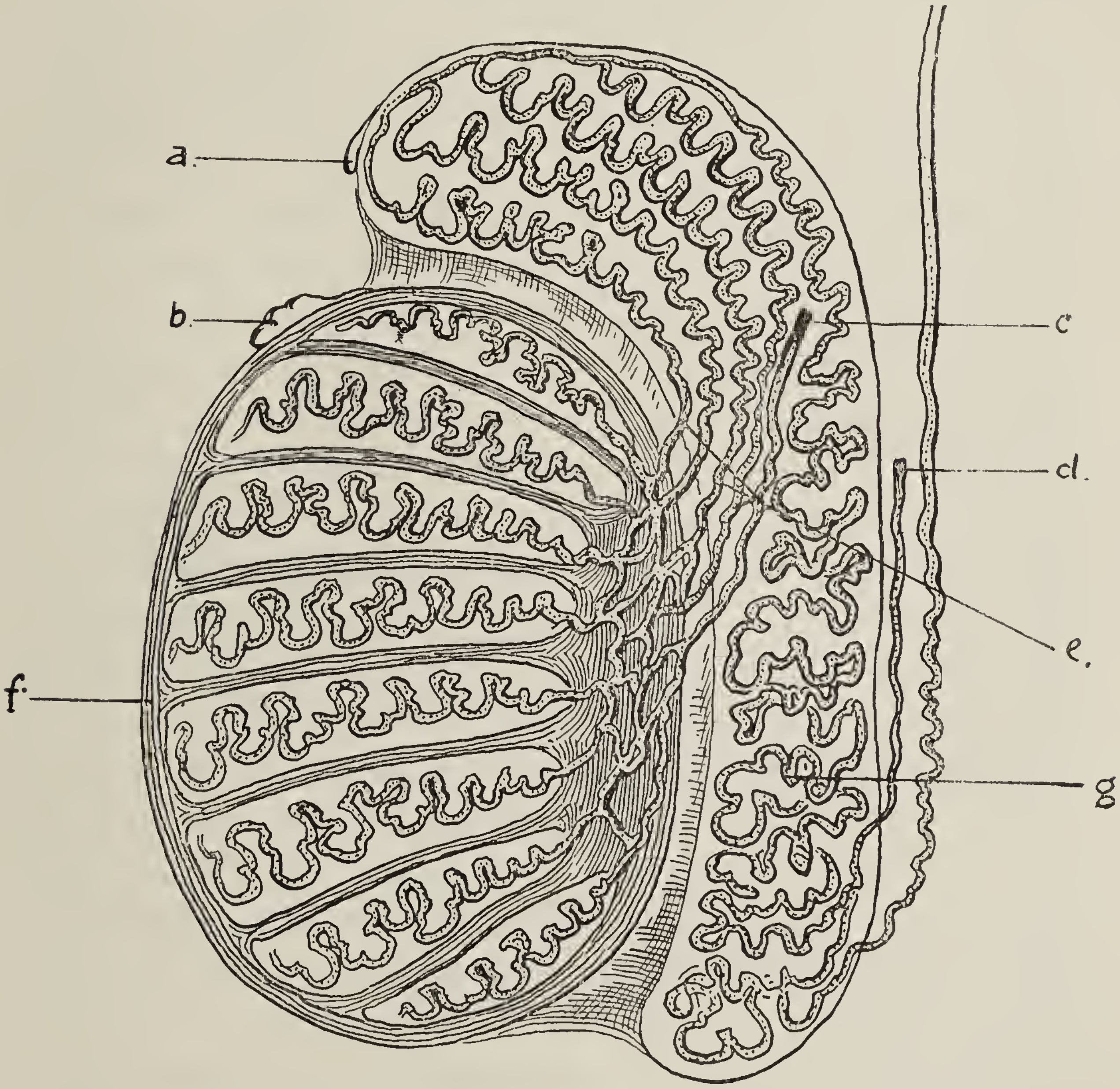

Fig. 161.—Diagram of the testicle. *a*, globus major and pedunculated hydatid; *b*, non-pedunculated hydatid; *c*, vas aberrans superius; *d*, vas aberrans inferius; *e*, vasa efferentia; *f*, tunica albuginea; *g*, globus minor.

The epididymis is attached to the testicle at three points by two-leafed folds of serous membrane, which are nothing more nor less than the extension of the visceral layer of the tunica vaginalis on to the epididymis. The fold at the globus major is called the ligamentum epididymidis superius and between its leaves pass the vasa efferentia. The double fold at the body encloses a space, saccus epididymidis. The ligamentum epididymidis inferius is the fold at the cauda.

There now remain to be considered the embryonic remnants, viz., the hydatid of Morgagni, the pedunculated hydatid, the vasa aberrantia and the organ of Giraldès.

The origin of the hydatid is still in dispute. Luschka claimed that the sessile hydatid arises from the upper Wolffian tubules and the pedunculated hydatid from the Müllerian ducts. Virchow and Kocher claimed that the sessile is a remnant of the Müllerian ducts. Stohr believed that the pedunculated is a representative of the Wolffian body.

The sessile hydatid is found at the upper pole of the testicle, close to the globus major, and it has been stated by Kocher, Hochenegg, Lewin and Luschka that there is a communication between the canal of the epididymis and hydatid. The pedunculated is attached to the globus major by a stalk; the stalk and head are usually solid. The vasa aberrantia are two in number, the superior and the inferior. The superior rises from the rete testis and ends blindly in the globus major; the inferior springs from the canal of the epididymis or from the duct of the vas deferens and ends blindly after extending up the cord about two inches. Kobelt proved that the inferior one was a remnant of the Wolffian body. Concerning the origin of the superior there is some doubt. Langer-Toldt looked on it as a vas efferens that had lost its connection with the epididymis.

The organ of Giraldès is a group of vesicles and tubules situated in front of the vessels of the cord at the height of the globus major.

Origin. Liston regarded the cysts from which he had withdrawn the fluid containing spermatozoa as enlarged seminal tubules.

Curling, Broca and Paget advanced the theory that these cysts were neoformations. Curling and Broca maintained that the presence of spermatozoa was due to a rupture of seminal tubules into the cyst. Paget thought that the spermatozoa were produced by the epithelium of the cyst.

Following this, Follen and Verneuil produced the idea that this peculiar condition was a remnant of the Wolffian body.

To Virchow is given the credit for the theory held today, namely, that spermatoceles are retention cysts; however, he believed that they arose only from the vasa efferentia and the organ of Giraldès.

Kocher contended that the constant site of the spermatocele was at the vasa efferentia. That the latter structures offer a favorable location was confirmed by Hochenegg, who found the width of the seminiferous tubules to be 0.1 to 0.2 mm.; that this diminishes to the size of the capillaries in the rete testis; in the vasa efferentia there is an expansion to 0.6 mm., followed by a contraction of 0.2 mm. in the coni vasculosi. This anatomic arrangement places the vasa efferentia between two obstructions, and as the testicle and globus major are covered with a dense capsule which would impede the formation of retention cysts, while the vasa efferentia are only surrounded by loose connective tissue, it is quite readily seen that dilatation of these ducts could easily occur. As a matter of fact, Dolbeau proved this experimentally by the injection of one hundred testicles with mercury, which caused a dilatation of the vasa efferentia.

The vas aberrans superius having an origin similar to the vasa efferentia, and since it has similar surroundings, together with the fact that it ends blindly, can be considered as a point predisposed to retention cysts. As for the vas aberrans inferius a retention cyst here would mean an obstruction of the duct of the vas.

If the hydatids or the organ of Giraldès are in communication with the semen-carrying system the origin of spermatocele from these areas is quite possible. However, since the organ of Giraldès and the pedunculated hydatids are usually isolated, not very much credence can be put into any theory, including these organs as the points of origin of spermatocele. From the clinical and anatomical data it seems probable that the sessile hydatid is often involved. Therefore in summing up it can be said that spermatoceles should arise most frequently from the vasa efferentia and vas aberrans superius, often from the sessile hydatid and only occasionally from the vas deferens, pedunculated hydatid and organ of Giraldès.

Causes. 1. *Trauma.* Gosselin was the first to give trauma as a cause, and later Hochenegg described the *modus operandi* of this factor. According to the latter, on violent concussion the testicle, which is more or less mobile, is able to descend, whereas the epididymis, which is fixed, cannot follow the motion, and as a result there is tension on the folds of the serosa, and as the tension increases the vasa efferentia are torn. This laceration, he states, usually occurs where the vasa efferentia pierce the tunica albuginea; the ends obliterate and blind pouches are formed. Another method is given by the same author, namely, that on account of the very free blood supply trauma may cause subserous bleeding, which becomes organized and converted into connective tissue, and as this shrinks later the vasa efferentia or the canal of the epididymis is obliterated.

In support of the trauma theory, Hochenegg reported two cases: one in a man, aged fifty-two years, in whom after a sudden fall a stinging pain in the groin was felt and followed six months later by spermatocele; and the other in a boy, aged fourteen years, who after lifting a heavy load had a history similar to the above.

Princeteau, Verneuil, Krebs and Bonneau reported spermatoceles following injury. In Bonneau's case it followed an operation for hydrocele. Krebs found trauma as a factor 8 times out of 15 cases.

2. *Sexual Abstemiousness.* Cavasse reported the first case that gave rise to this theory, but it has neither strong clinical nor pathologic support.

3. *Gonorrhea.* It is to be expected, of course, that to venereal disease would be ascribed a role in this condition.

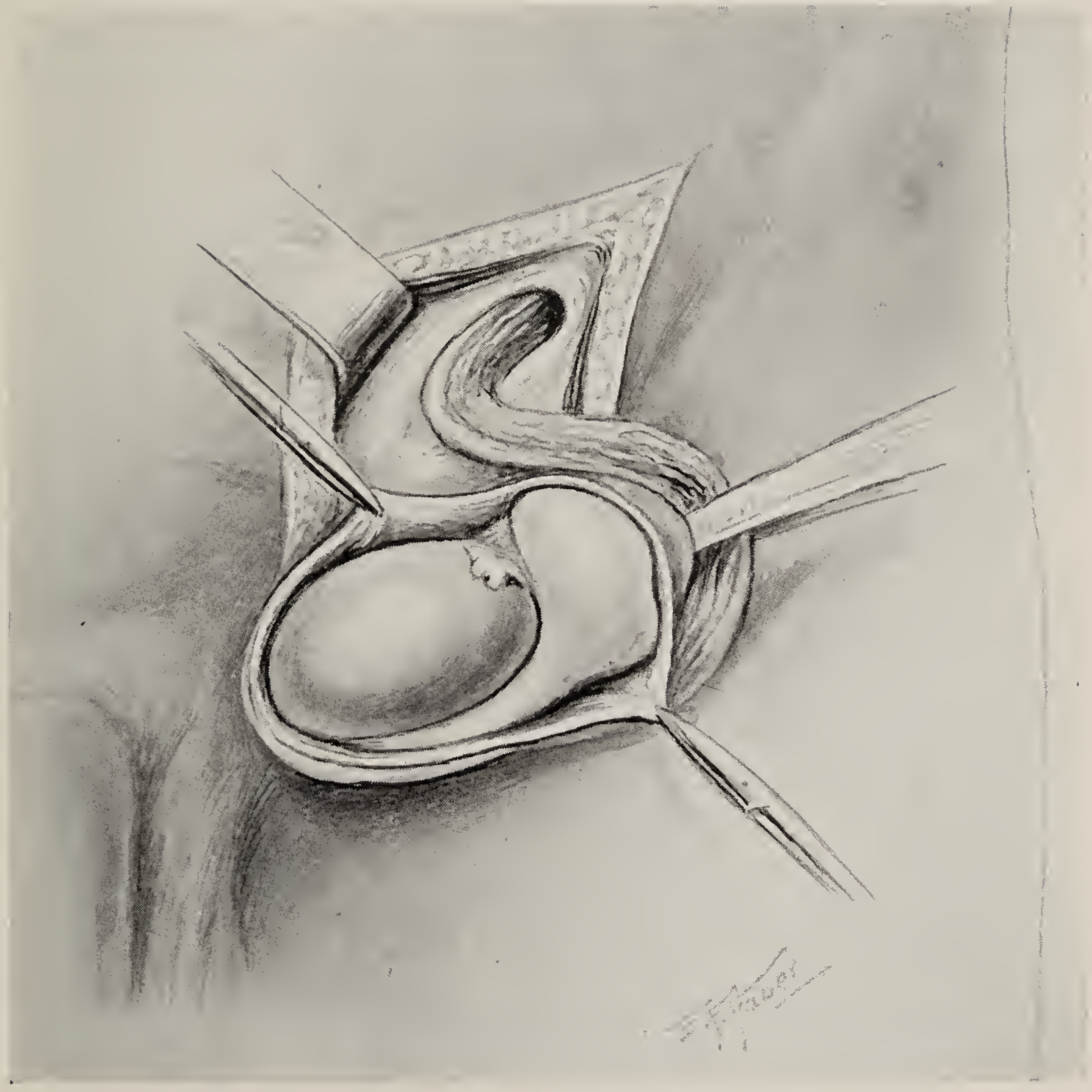

FIG. 162.—Case of spermatocele. Note the cyst developed at the globus major.

However, there is very little clinical evidence to support it, and yet why should it not be, particularly when the epididymis is involved?

Of the causes above named, trauma seems to be the most logical one, yet it must be of a peculiar variety, for injury to the scrotum is not rare while spermatoceles are a curiosity. As before mentioned it is quite logical to expect that gonorrhea might be an etiologic factor; however, one cannot understand in that event why spermatoceles should not be as numerous as cases of hernia. In sexual abstemiousness the overflow finds its way first to the seminal vesicles and then gains exit through pollutions instead of forming retention cysts.

Evidence has been produced to prove that spermatoceles are more numerous than suspected. Lewin found 8 of these cysts in 100 testicles of cadavers, while Hochenegg found 27 in 332 testicles of cadavers.

In the cases reported the incidence seems to be equal on the two sides. They may occur at any age after the testicle has commenced to function.

CLASSIFICATION. Von Hoffman divided them into intravaginal and extravaginal, though he claims that most of the extravaginal cysts commence intravaginally.

Extravaginal. This variety arises from the vas aberrans superius, from the vasa efferentia and very rarely from the vas deferens and the paradidymis.

Those from the vas deferens and paradidymis are primarily extravaginal, whereas those from the other two, according to von Hoffman, become so by first occupying all the space in the ligamentum epididymidis superius, thereby displacing the epididymis backward, and then they grow upward in the tissues of the cord, when they become extravaginal. In this latter type of cyst the testicle is displaced downward and forward; the position of the testicle is unchanged in the variety arising from the vas deferens and the organ of Giraldès.

The site of insertion of the cysts arising from the vasa efferentia and the vas aberrans superius is in the rete testis; from the vas deferens and the paradidymis in the cord.

The extravaginal cysts are usually unilocular; if multilocular ones are found they are probably due to several cysts arising at the same time. The extravaginal cysts are larger than the intravaginal, and in one case reported by Stanley forty-nine ounces were removed at the first tapping and fifty-seven at the second.

These cysts are supposed to have a pear shape, which when seen is characteristic, but the larger cysts take almost any form. When seen macroscopically it is noted that the walls of the cysts are thin and that distributed in the sac walls are fibrous cords, which when the sac is greatly distended cause bulgings, giving the balloon shape described by Savariaud.

Intravaginal. These arise from the sessile hydatid or the canal of the epididymis, and project into the cavum vaginale. That these cysts do occur at the hydatid of Morgagni was demonstrated by Hanusa, who found during an operation for hydrocele a dilated sessile hydatid the size of a cherry and containing spermatozoa. As the walls of the hydatid are very thin, rupture into the cavum vaginale can easily occur, giving rise to a hydrocele containing spermatozoa.

The intravaginal cysts become intimately connected with the tunica albuginea, and as they grow they take on the form of the globus major. It is this latter characteristic that gives rise to the impression that there is an accessory testicle superimposed on the normal organ.

PATHOLOGY. *Structure.* The walls of the cyst are made up of interwoven strands of connective tissue, among which can be found bundles of smooth muscle. These bundles arise from the smooth muscle of the vasa efferentia and the canalis epididymidis, and when found, it is claimed, they surely point to the presence of spermatocele. The cysts are lined with ciliated or cylindric epithelium when recent; in the older varieties the lining is of pavement epithelium.

Contents. The contents are usually milky or soapy in color. On microscopic examination, lymphocytes, fat globules, epithelial cells and spermatozoa are found. The spermatozoa may be rigid or mobile, and in the cysts arising from a testicle which has become senile the spermatozoa may be absent. The fluid is feebly alkaline, has a specific gravity of 1.002 to 1.006 and contains 0.2 to 0.5 per cent. albumin.

Symptoms. Subjective. There is a group of symptoms which, though rarely found, is pathognomonic, namely, severe pain in the testicle and swelling on sexual excitation. In the majority of cases there is no pain, only a feeling of distress. The swelling or tumor is what is most complained of, and if this is a slowly progressing affair following trauma it would cause one to suspect spermatocele. A certain number consult a physician, believing they have a third testicle, and as Liston says, "flatter themselves in thinking they are thus unduly provided."

Objective. Here too the only finding will be a tumor. In the intravaginal variety it is a globular mass sitting on top of the testicle, but, as sometimes occurs in this variety, a hydrocele may be coexistent and the tumor will not be recognized.

In the extravaginal cysts the testicle will be found displaced downward and forward.

DIAGNOSIS. The history of a trauma, followed by a slowly progressive tumor in the scrotum, would be circumstantial evidence. If a pear-shaped mass is found with the testicle displaced downward and forward the diagnosis of extravaginal spermatocele is probably correct, as is also the finding of a globular mass on the top of the testicle, indicative of the intravaginal cyst. Presence of spermatozoa in the aspirated fluid is not a positive proof, for hydrocele may contain these elements.

Hochenegg resorted to chemical examination for differentiation of hydrocele and spermatocele fluid; spermatocele fluid, as stated above, is feebly alkaline, has a specific gravity of 1.002 to 1.006 and contains 0.2 to 0.5 per cent. of albumin, while hydrocele fluid is strongly alkaline, has a specific gravity of 1.020 to 1.040 and has from 4.4 to 7 per cent. albumin. No dependence can be put on the light test, as spermatoceles transmit light as well as hydroceles.

Treatment. Aspiration, injection of irritants and incision have all been abandoned, as recurrences were frequent.

The radical operation is now performed. In the cysts arising from the sessile hydatid and the vasa efferentia a portion of the tunica albuginea must be removed and the defects covered with serosa. In the other varieties the cyst can be easily enucleated from its bed and the stalk ligated close to its base. If the testicle is atrophied castration is the logical procedure.

Case Report. L. O., aged eighteen years, male, white, admitted to the Protestant Episcopal Hospital September 12, 1916 and discharged September 30, 1916.

Family History. Negative.

Past Medical History. Has not been ill since childhood. Denies venereal infection.

History of Present Illness. For the past six years the patient has noticed sagging in the left scrotum, and for about the same period has noticed a tumor attached to the testicle. At first it was the size of a pea, now had grown to the size of a walnut. On account of the tumor the patient was given the nickname of "Hock shop." Never any pain in the testicle nor any interference with genito-urinary functions. Comes to the hospital to be operated on, as he wishes to enter the navy, and has been rejected because the navy doctors said he had a "third testicle."

Physical Examination. Head, neck, chest and abdomen negative.

Scrotum. Varicocele on left side. Attached to the upper pole of the left testicle, at the site of the globus major, is a mass the size of a walnut. It is firmly attached to the testicle and pressure over the mass is transmitted to the testicle. A diagnosis of spermatocele was made.

Operation (September 15, 1916). Operator: Dr. A. P. C. Ashhurst. Assistant: Dr. Crossan. Incision over the left inguinal canal, three inches long. Fascia of the external oblique is split and the testicle delivered into the wound by traction of the cord. Tunica vaginalis seemed to be slightly distended and did not show any masses on the external surface. Tunica vaginalis is then opened and a few drams of clear straw-colored fluid evacuated. Tunica is split throughout its whole length, and it was then ascertained that the globus major showed a cyst the size of a lima bean. This was aspirated and about 4 c.c. of a milky fluid removed. The walls of the cyst were then opened. On opening the wall of the globus major it was found there was another cyst wall inside of it at a depth of 0.5 cm. This was dissected free and removed having no dense adhesions. Portion of the wall of the globus major is removed for examination. Remainder is closed with No. 00 chromic catgut. Previous to the above procedure a small portion of the tunica vaginalis was removed at the upper angle. The tunica vaginalis is then closed with No. 0 chromic interrupted gut. It was impossible, however, to make the edges meet at the upper limit of the sac. Testicle and its covering are replaced into the scrotum. Varicose veins separated from the remainder of the cord and divided between ligatures, a length of about 6 cm. being excised, and stumps approximated by tying the corresponding ends of the ligatures. External oblique is then closed by continuous No. 1 chromic catgut. The skin with No. 0 lock stitch of chromic gut. Dressings are then applied.

September 30. Discharged. Testicle is still swollen and tender on pressure.

Pathologic Examination. 1. Cyst Wall: Fibrous tissue wall, lined with columnar cells.

2. *Fluid from Cyst:* Two c.c. in amount, contained two spermatozoa, specific gravity 1.050.

Further History. Patient was seen on March 2, 1920, at which time it was found that there was an atrophy of the left testicle. The latter was reduced to a mass 1½ cm. long by 2 cm. wide. The above unfortunate occurrence was probably due to some impediment at the globus major, completely obstructing the semen-carrying system. Briaud produced a similar condition in rabbits by ligating the vas deferens.

SUMMARY. Spermatocele is a retention cyst of the scrotum which is or has been in communication with the semen-carrying system. The cysts arise most frequently from the vasa efferentia, the vas aberrans superius and the sessile hydatid. The main symptom is swelling, with sometimes a history of previous trauma. The treatment is extirpation.

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CESAREAN SECTION IN THE TREATMENT OF ECLAMPSIA[1]

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THE obstetrician is confronted with few situations more appalling than that of an expectant mother with convulsions, where the life and well-being at once of two patients is suddenly thrust upon his care. That the cases of this kind coming under the notice of a single practitioner are happily few does not render the management of them any easier. Indeed, their rarity is more likely to lead to unpreparedness. These are cases where resourcefulness, prompt and often radical measures determine whether the end result will be the recovery or the loss of one or both patients.

Inasmuch as the real cause of the condition is not known specific treatment based on such knowledge cannot be applied. Hence our treatment becomes empiric and differs very materially with different authors. That it is due to a toxemia is universally admitted; pathologic changes in the kidney, liver and brain as a result of this toxemia are well recognized; and that pregnancy is responsible for the condition is conceded, although the precise laboratory identification of the poison and its nature are matters of controversy. Given these premises, treatment directed to the elimination of the poison legitimately follows. Thus far, all are in accord. At this point begins the debate as to conservatism or radicalism.

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[1] Read at a meeting of the Philadelphia Obstetrical Society, February 7, 1918. Reprinted from the American Journal of Obstetrics and Diseases of Women and Children, 1918, No. 6, vol. lxxvii.

There are in vogue at this time two distinct and divergent plans of treatment of antepartum eclampsia. The one, that of the Rotunda and Stroganoff schools, which addresses its treatment to the convulsions and lets what may happen to the pregnancy. The other, the more widely adopted plan, proceeds promptly and boldly to get rid of the cause by emptying the uterus and then follows this up by eliminative treatment. The latter appears to me to present the more convincing argument; for, as Peterson who has written much about this subject, says: "There is no gainsaying the fact that the presence of the fetus is the underlying cause of the eclamptic convulsion of the mother, whether eventually it be proved that eclampsia be of maternal or of fetal origin. Postpartum eclampsia, to be sure, occurs with the uterus empty but it is the result of an intoxication brought about by a pregnant uterus, not lessened sufficiently by the emptying of that uterus to prevent the eclamptic convulsive explosion during the puerperium. Every advocate of the medicinal or so-called conservative treatment of eclampsia would be much better pleased when confronted with a case of antepartum eclampsia if the uterus were soon to be emptied."

Happily, the uterus often empties itself spontaneously during the course of the eliminative treatment and thus solves the problem which the advocates of immediate delivery aggressively undertake, but to depend upon the efforts of elimination without cutting off the source of the poison would seem to be on a parity with the endeavor to build merchant marine vessels to relieve the food situation in Europe faster than the U-boats can destroy them.

If one adopts the practice of prompt delivery on the occurrence of a convulsion, it is important to have well thought out methods for accomplishing this and to know the limitations of each, for the suddenness of the onset leaves little time for deliberation and every hour lost in expectant treatment is a golden one. Statistics show that the more promptly the delivery is accomplished after the onset, the better is the prognosis. To quote again from Peterson: "Olin, who from the nature of his statistics was able to estimate accurately the time elapsing between the first convulsion and the operative delivery of his patient, found the mortality in thirty-one cases, delivered one to three hours after the onset of the first convulsion, to be only 3 per cent.; on the other hand, in 50 cases delivered from six to twenty-four hours after the first convulsion, the mortality was 28 per cent." This is in harmony with the conduct of other grave disorders, as, for instance, herniæ, perforations, appendicitis, etc., and is only common sense. Delivery should therefore be undertaken at the earliest possible moment after the first convulsion.

The methods of quick delivery are practically limited to three:

1. Dilatation of the birth canal and the application of forceps, or version, called by the French, "*accouchement forcé.*"
2. The vaginal Cesarean operation.
3. The abdominal Cesarean operation.

It is to the latter procedure that this report is a contribution. Each case should be considered individually and that method adopted which best suits the case.

In what class of cases then would one elect to do an abdominal Cesarean operation? Obviously, in cases of disproportion, where the indication is absolute, and in the presence of tumors or deformities which obstruct the birth canal. These cases call for abdominal section whether complicated or not by the occurrence of eclampsia. If the obstruction is not an insuperable one, but merely the normal undilated natural birth canal of a primipara, is it not possible to deliver the woman more speedily, with less injury to the mother and none at all to the child and with less exposure to septic infection through a clean abdominal incision than through a prolonged and tiresome dilatation of an unobliterated cervix, through a canal which can scarcely remain sterile throughout its manipulation and which invariably suffers trauma to a greater or lesser degree and if the trauma extends up through the cervix to the base of the broad ligaments it is of a type that cannot be satisfactorily repaired. I believe that we may confidently give an affirmative answer to these queries.

Does shock play an important part in the progress of the case, and is it more marked after a section than after *accouchement forcé?* Of course, severe shock is a serious matter in any surgical condition, but it is my impression that shock is not more marked after a section than after a forced delivery through the natural passages in this special group of cases. Indeed, I suspect that a mild degree of shock is of benefit in arresting the convulsions. Surely the bleeding which results from operation is indicated, for withdrawing blood is the most important single procedure in the management of hypertension, and the amount of blood lost in the course of a Cesarean operation should not exceed the amount purposely withdrawn by phlebotomy. Moreover, the surgical bleeding from a section is under better control than that which arises from injury to the lower uterine segment, the vault of the vagina and the cellular tissue about the cervix, which almost invariably results from forced delivery.

Those of you who have had much experience in dragging a baby through the undilated birth canal have, I am sure, often felt chagrined and disappointed at the end-result. It is an operation which requires skill, infinite patience and much time, much more time, indeed, than one likes to keep a patient under an anesthetic. This statement applies, of course, to the primiparous patient, not in labor.

Let us for a moment consider the contra-indications for an abdominal Cesarean operation. The Cesarean operation in its early history was undertaken only as a last resort and until less than ten years ago, for some obstructive reason, where for instance, trial labor had failed; the consequence was that the mortality was high. It required a long time to learn that the gravity of the operation arose chiefly from infection due to repeated vaginal examinations and more particularly where the bag of waters was ruptured. There is nothing about this operation itself to make it more serious when there is no infection present than is celiotomy for any other purpose. Infection is the important point to bear in mind. How shall we avoid this? The answer is plain—avoid vaginal examinations. You must have observed how well your patients get along who have their babies before you arrive on the scene. This should have furnished food for thought. For about two years it has been the rule at the Kensington Hospital for Women to make no vaginal examinations upon patients entering into labor. Obtainable information is to be gotten in the first place by examination of the abdomen, by palpation and auscultation; and in the second place by rectal and not by vaginal examination. Thus, in a very high percentage of our cases, we are able to conduct the whole process of parturition without a single vaginal examination, and we feel that our temperature charts reflect the advantages of this procedure. With this routine, we do not have the slight temperature curve on the second, third or fourth day, which we used to call "milk fever," because it was about synchronous with the inflow of milk into the breasts. The gain is not alone to the mother but to the doctor as well, for the accepted routine heretofore has called for a thorough scrubbing of the hands, the use of some antiseptic solution and a sterile glove before an examination should be made, all of which was a time-consuming, and in cold weather a skin-destroying process. Now we walk in, put on any old glove, without hand preparation, apply some lubricant, protect the vulva with a sterile towel or gauze and introduce the finger into the rectum, and what do we learn? This is the question that each new resident puts to us. There is no difficulty in determining whether the presenting part is high up or low down in the pelvis. If the cervix is unobliterated it is easily felt through the bowel; if it is flattened and partly dilated it presents a more difficult problem. But even so, patience and perseverance will usually yield the answer to an experienced examiner, who feels around for the thinnest place between the examining finger and the presenting part and is then able to hook up at one point or another a margin of the os and so to follow it around and determine the degree of dilatation. The direction of the sagittal suture can be recognized even though there be scant or no dilatation. When this information is supplemented by that gained from an external examination we have all the needed facts in the great majority of the cases. I grant that skill in this matter arises only from practice, but it is worth it. One's first efforts are discouraging but persistence will be rewarded. If it is determined that the case is exceptional and some operative interference is called for, then, of course, a vaginal examination is made, and with the same antiseptic precautions that are observed when instrumental delivery by the natural route is undertaken. Statistics can be adduced to show that repeated vaginal examinations, made hours before delivery, give rise to a higher percentage of infections than examinations made shortly before delivery, thus fortifying the practice of deferring vaginal examination until artificial aid is invoked.

The first questions asked when a patient with convulsions is admitted to the hospital are: "Has she had repeated vaginal examinations and over how long a period? Is the bag of waters broken and have there been futile efforts at delivery?" The answer to these questions determines for or against a Cesarean method of delivery in a group of cases where this method may be employed. This group, as has already been intimated, consists of women pregnant at or near term who have never given birth to a baby, and hence have undilated birth canals. Rarely multiparæ, with rigid and high-placed cervices, with the baby well into the viable period, and the head not fixed in the pelvis may be included in this group. To these may be added, to wit: Those with relative disproportion, tumors, scars, ulcers, infected wounds of the soft parts, marked edema of the vulva, and last, but not least, subjects of a previous ventrofixation, for of course, all these can become victims of eclampsia.

In the modern consideration of the Cesarean operation, for whatever purpose, the rights of the unborn child assume an increasing importance. There are few circumstances, for instance, where one would resort to this procedure to deliver a dead baby. However, the interest of the mother is paramount and for this particular disorder the operation is undertaken almost exclusively in her interest, for the babies too often are poor risks, either on account of prematurity or toxemia. In a case seen recently by me, where the baby died, the manufacture of toxins seemed to be arrested and I believe this is the rule. Induction of labor led to the birth of a dead and macerated fetus. These babies participate in the toxemia of the mother and naturally the longer they are exposed to the poison the more saturated they become with it. There is no accurate way of measuring the toxicity of the mothers' blood, for sometimes they die after a single convulsion and often survive many convulsions, but this statement is probably true —that the more severe and the more frequent the convulsions the greater the toxemia. Therefore, when the child is alive and near term the clearer is the indication for early operation. Furthermore, the interest of both the mother and the child lie in the same direction, that is, in early operation, for by this means we cut off the further manufacture of toxins and thus, as it were, destroy the enemy at its base. So far as the interests of the child are concerned, if it be viable they are far better served by abdominal delivery than by any other method, for it is thus spared the trauma and shock of a tedious birth through the natural passages, not to mention the prolonged exposure to the anesthesia of the mother.

Another and important consideration is the environment of the patient and the experience and the skill of the operator. The operation itself is among the simplest of major surgery but should not be undertaken by anyone untrained in antiseptic technic and without some experience in abdominal surgery, nor should it be undertaken without competent help, for there is no doubt that there is more risk in invading the abdominal cavity than in operating through the vaginal canal, under a careless technic. Where a hospital with modern equipment is available it is better to transport the patient to the hospital than to make the needed preparations for a section at home, for the indication for the operation arises so unexpectedly that the household is totally unprepared and the time required to assemble the assistants and prepare for the operation at home would be longer than it would take to carry the patient to the hospital where help and facilities for operating are at hand; thus a hospital equipment or its equivalent is an important factor in selecting the abdominal route for delivery.

Following is a report of 21 cases of eclampsia delivered by abdominal section, in addition to or preliminary to eliminative treatment. Two of these mothers died (and four babies), a mortality of 10 per cent. These cases were not selected because they appeared to be favorable, but were cases good and bad as they presented themselves under the conditions named above. Many of them were not early cases; that is, the operation was not done promptly after the first convulsion which is the time of election, but even so the percentage of recoveries compares not unfavorably with that of other methods of treatment. However, I desire only to record these cases and not particularly to lay stress upon their percentage value, for of course such a small number has no value except as a contribution to a larger series.

CASE I.—C. B., Polish, primipara; aged twenty-nine years; admitted to Protestant Episcopal Hospital November 7, 1911, in a comatose condition. Venesection and medical treatment were instituted and in spite of this convulsions continued, one of them, according to the notes, was practically continuous and lasted thirty minutes, at the end of which time she was so swollen and cyanotic that it appeared as if she were about to die. I had never at this date done a section for eclampsia. The baby's heart sounds were audible and to attempt a delivery through the undilated and edematous natural passages of the mother, who appeared to be moribund, seemed futile, so a section was decided upon with a view of rescuing the baby, and to my amazement both the mother and child survived the operation. After two or three days of profound coma consciousness began to return, but more amazing was the changed appearance of the individual, from a bloated, edematous, cyanotic mass of flesh, the features began to dawn and the limbs to assume a natural shape, and by the end of ten days or two weeks there appeared an individual seemingly less than half the size of the one operated upon. The baby, which was well developed and healthy in appearance at first, failed to grow, although taking its nourishment well and apparently digesting it, being fed artificially, of course. It grew more and more leaden in hue and died after thirteen weeks, weighing the same as when it was born.

Case II.—E. H., aged nineteen years, admitted to Protestant Episcopal Hospital February 7, 1914, eight and a half months pregnant. Bowels and kidneys have been regular and active. No history of headaches. The onset of the illness was sudden. She was brought to the hospital in the ambulance in convulsions, pulse 138, temperature 100.2°. Section was done on account of the great edema of the vulva and the undilated birth canal of a primipara, resulting in recovery of the mother and a living child.

Case III.—Mrs. V., aged thirty years, admitted to the Frankford Hospital. Her first baby was delivered by a mutilating process on account of disproportion. Now in her second pregnancy it was estimated that she was two weeks overdue without engagement of the head. Her legs, hands and feet were swollen, she had headache, and the urine contained albumin and casts. Section was decided upon for her delivery. Before she was brought for operation she developed a convulsion. Section resulted in a living baby and an uninterrupted recovery of the mother.

Case IV.—S. S., aged twenty years; primipara; admitted to Protestant Episcopal Hospital February 25, 1914, pregnant at term and in labor, with unruptured bag of waters. Had had seven convulsions before admission. Section was done on account of eclampsia and primipara and unruptured bag of waters. Result: Recovery of mother and stillborn child. No convulsions followed operation.

Case V.—B. W., aged twenty-three years; primipara; admitted to Protestant Episcopal Hospital February 26, 1915. Convulsions before and after admission. Pregnant eight months. Section resulting in recovery of mother and living baby. One convulsion followed delivery.

Case VI.—H. S., aged eighteen years, admitted to Protestant Episcopal Hospital April 29, 1915, primipara, after having an unknown number of convulsions. Section resulting in recovery of mother and a living baby.

CASE VII.—M. R., aged twenty-four years; primipara; admitted to Protestant Episcopal Hospital March 6, 1912. On March 2, complained of headache and dimness of vision. Had first convulsion on March 4, and was brought to the hospital on March 6, and eliminative treatment started, expectant treatment. However, she was operated on later the same day and died at 2 P.M. on March 8, 1912. The baby lived.

CASE VIII.—Mrs. D., nephritis, with marked edema and cough. Toxemia. Section resulting in living baby and recovery of the mother. Was admitted to the Kensington Hospital for Women on March 11, 1914, with a blood-pressure of about 200 mm., extensive edema of the legs and face, skin of marble hue, mental dulness, headache and scanty urine containing albumin and numerous granular casts. Her condition improved considerably after one week by the use of a milk and buttermilk diet, purgation and baths. The urine, however, continued scanty, the twenty-four-hour output for the 15th, 16th, 17th and 18th being 12, 25, 15 and 26 ounces respectively, and after a distinct gain for the first few days after admission to the hospital, she began again to lose ground even under strict treatment, so on the 19th it was decided to bring about delivery, even though no convulsions had occurred. Inasmuch as she was a primipara with breech presentation, abdominal section was elected in preference to induction of labor through an undilated birth canal. Owing to her cough chloroform was employed as an anesthetic and a 10-pound baby quickly delivered. After the uterus was emptied it was observed that there was a marked projection of the sacral promontory which would have greatly increased the difficulty of delivering a 10-pound baby presenting by the breech through the natural passages. Her operative recovery was uneventful.

CASE IX.—Nephritis; cardiac dilatation; edema of the lungs. Section: Followed by death of the mother and baby.

Mrs. G., aged twenty-four years; primipara; admitted to the Protestant Episcopal Hospital March 10, 1914, and died March 12, 1914, pregnant eight months. Has suffered a great deal throughout her gestation from headaches and constipation. Ankles and eyelids are edematous. For the past three weeks has been very short of breath, with cough, and expectoration of a mucoid material. This morning she grew suddenly worse. She could not lie down. She became deeply cyanotic and was constantly coughing up a frothy and bloody mucus, which was her condition on admission. The respirations were rapid and labored; the vessels of the neck engorged; the pulse very rapid, irregular and weak; the right heart was dilated and moist rales were audible all over the chest. Twenty ounces of blood was withdrawn at once from the median basilic vein and atropin, grain $\frac{1}{150}$, strychnin, grain $\frac{1}{30}$, and camphorated oil, ♏xx given hypodermatically, followed by some relief. Prompt delivery of the baby was regarded as imperative, and owing to the primiparity of the mother, section was decided upon as the quickest method of delivery. Morphin, grain $\frac{1}{4}$, was given hypodermically and the section done under local anesthesia, for which Schleich's solution was employed. The abdominal incision only required an anesthetic. For the uterine incision no anesthetic was used or needed. A well-developed baby, with pulsating cord, was quickly delivered, but the assistants, in spite of faithful efforts, could not succeed in establishing respiratory action. A large quantity of serum escaped from the baby's mouth and nose during the efforts to induce respiration, showing that it is probable that this infant sucked amniotic fluid into the bronchial tubes by intra-uterine respiratory efforts, stimulated by the blood highly charged with carbon dioxide which was furnished to it. Thus the baby appeared to be literally drowned in amniotic fluid. Following the operation, which was done about midday, the patient was quite comfortable, with improved pulse, able to lie flat and relatively free from respiratory difficulty. Although pallid and waxy in appearance her improved condition seemed to justify the hope that she might recover. She, however, passed only 2 ounces of urine during the first eighteen hours after operation, but 17 ounces were passed during the next eighteen hours. The abdomen became more and more bloated, the bowels resisting all measures for their evacuation. Toward evening nausea supervened, for which the resident physician passed the stomach tube, whereupon the patient had a convulsion and died about thirty-six hours after delivery. Her urine boiled solid with albumin and was loaded with granular casts.

Case X.—Mrs. R., aged nineteen years, admitted to the Kensington Hospital for Women August 24, 1915, primipara, apparently at term. Two convulsions had occurred before admission and one after admission to the hospital. Section was promptly resorted to and a living child recovered, which lived only one day. Mother was discharged as cured on the sixteenth day.

Case XI.—Mrs. W., aged twenty years, admitted to the Kensington Hospital for Women January 22, 1916. Primipara; overdue; presenting part not engaged; marked edema of the legs; R. O. P. Urine boiled solid; many light and dark granular casts; systolic blood-pressure, 185. Delivery was decided upon and abdominal route selected and a baby weighing 6 pounds 3 ounces recovered. Mother and baby discharged well on the fifteenth day.

Case XII.—Mrs. L., primipara; aged twenty-one years, admitted to the hospital March 15, 1916; having had four convulsions since 7 A.M. and being about seven months pregnant. At about 2 P.M. Cesarean section was performed and a live but dyspneic baby recovered. The mother had been given morphin sulphate, grains 1¼, from 9 to 1 P.M. The baby lived about twenty hours, having had two or three convulsions and perhaps dying in a convulsion. The mother recovered.

Case XIII.—Mrs. S., admitted to the Protestant Episcopal Hospital July 9, 1915, with a diagnosis of toxemia of pregnancy, and medical treatment given, in spite of which the toxemia deepened. She was about seven months pregnant, hence it was decided that the cause of the trouble should be removed. Section was adopted as the method of choice on account of the primiparity. A live baby weighing two pounds was secured. The mother recovered.

Case XIV.—Mrs. B., admitted to the Kensington Hospital for Women, March 29, 1916. Had eight convulsions with first pregnancy and was delivered at the Protestant Episcopal Hospital by forceps of a dead baby on account of disproportion. This time her legs, hands, and face were much swollen, and she had loss of sight for eight hours. The bag of waters broke and she had a show three weeks before operation and the baby was supposed to be two weeks overdue. No convulsions had occurred. However, operation was done and a 7-pound 10-ounce baby secured. Mother and baby made a good convalescence. Mother lost 28 pounds in weight in the twelve days following the operation, most of which must have been water.

Case XV.—Mrs. R. I., aged nineteen years; primipara. Has suffered with edema of feet and headache for the past three months. At 2 P.M. September 13, 1916, had her first convulsion. Before her admission to the hospital at 8 P.M. she had three more. Blood-pressure on admission was 190–150, and after admission four more convulsions at short intervals before being brought to the operating table. Operation resulted in a living baby, weighing 5 pounds 8 ounces. Patient passed a very restless night and after eighteen hours treatment showed marked improvement. Result, recovery of mother and baby.

Case XVI.—Mrs. G., aged twenty years; primipara, brought in in the ambulance, having had several convulsions. Blood-pressure on admission 170–110. Three convulsions after admission.

Patient maniacal between convulsions. Operation resulted in a 6-pound 11-ounce baby and recovery of mother.

CASE XVII.—Mrs. E., aged twenty-one years; primipara. Has suffered with headache for three months and edema. Had several convulsions at home. Brought to the hospital in the ambulance. One convulsion while being prepared for operation. Operation resulted in an 8½-pound living baby. Result, recovery of mother and baby.

CASE XVIII.—L. K., aged twenty-three years; primipara. Was well until one week before admission when she complained of edema and headache. Was brought in by the ambulance. One convulsion in the ambulance and four previously. Operation resulted in a 7-pound 4-ounce living baby and febrile recovery of the mother.

CASE XIX.—E. B., aged thirty-five years. Was first admitted to the Kensington Hospital for Women on August 21, 1917, after having had three convulsions. Systolic blood-pressure on admission was about 96, and even after the convulsions there was only a trace of albumin in the urine and very few casts. She was unconscious. She was given at once intravenously a solution of sodium bicarbonate and glucose and purged, and after about twelve hours became conscious and after a few days returned home and was directed to take purgative medicine every morning and live on milk. She continued well for some days. She then developed a pain in the upper abdomen and right chest and thought she was in labor. The following twenty-four hours she passed very little urine and had some headache, but practically no edema. She was then re-admitted to the hospital and as convulsions were thought to be again imminent, abdominal Cesarean section was done and a live baby weighing 5½ pounds was delivered. The baby cried lustily immediately after extraction. It was put in an incubator, but lived only a few hours. Mother made a good obstetric recovery, but developed pleuropneumonia about ten days after returning home and was admitted to another hospital.

CASE XX.—J. P., admitted to the Kensington Hospital for Women on November 14, 1917, having had ten convulsions before being brought to the operating table, which occurred fifteen hours after the first convulsion. The patient was in labor on admission but the cervix was high up and undilated. A baby weighing 8 pounds 2 ounces was secured. Both the baby and mother made a good recovery.

CASE XXI.—G. H. L., aged twenty-one, primipara. Increasing toxemia while under treatment. Systolic blood-pressure on admission was 180. After a cabinet sweat the blood-pressure was 198, associated with ringing in the ears and intense headache and some edema. Delivery was decided upon, and owing to the primiparity and the undilated birth canal, section was resorted to. Result, recovery of mother and baby. Patient discharged in two weeks.

CONCLUSIONS. This argument and experience seem to justify the conclusions which may be briefly summarized as follows:

1. Since pregnancy is responsible for the eclampsia, its termination by one method or another is indicated as soon as a convulsion has occurred.

2. The interest of both mother and child are served by the same procedure—namely—early operation.

3. Each case should be considered on its own merits and that method of delivery adopted which best suits the case.

4. Delivery by the abdominal route is indicated in primiparæ where labor has not set in—or is not well advanced—where the child is living and viable, and the nearer the child is to term, the stronger the indication.

Multiparæ at or near term—but not in labor—with rigid and high placed cervices, may well be included in this group.

5. The contra-indications for the Cesarean operations are repeated vaginal examinations, examinations continued over a long period, more particularly where the bag of waters has been broken; and, of course, where unsuccessful attempts at delivery have been made, and where there is any infective process in the birth canal.

6. Operations through the abdomen are attended with more risk than operations through the birth canal if done under unfavorable conditions, hence a suitable environment and a capable operator and assistants are a *sine qua non* for this method of delivery.

OPERATIVE TECHNIC OF THERAPEUTIC ABORTION[1]

By WILLIAM E. PARKE, M.D.
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The conditions for which a therapeutic abortion may be justified will arise at any time after the first month following conception, to the date of normal delivery, and the method of accomplishing it will be vastly different in the early and late periods. Broadly speaking the procedure of dilatation and curettement is applicable to pregnancies of four months and less, while beyond this period, owing to the great liability to hemorrhage, the process of inducing labor should generally be resorted to.

The use of drugs of the oxytocic class is not worth while. They are too unreliable to depend upon. The use of tents, or indeed of any slow method is attended with so great a risk of infection as to preclude their adoption.

The method of choice in the early cases is that of the ordinary dilatation and curettement, bearing in mind at the same time that the operation should be performed with more delicacy than is necessary in dealing with the unimpregnated uterus, for the organ is both thinner walled and softer than is the normal uterus. Hence there is great liability to perforation. The patient should be anesthetized and the cleansing process conducted with as much scrupulous care as for a major operation. With the patient in the lithotomy position, the

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cervix, exposed by a self-retaining speculum, is seized and drawn down by a volsellum forceps; dilators (preferably the Goodell type) are introduced beyond the internal os and gentle pressure on the handles employed, the muscle sense guiding one as to how much pressure to make, until the canal is sufficiently open to admit a large sized curette and the curette forceps. This instrument I find indispensable, but it must be used with great skill, for it is easily capable of doing mischief. With it I seize the broken up fragments of the product of conception and extract them through the relatively narrow canal. Experience teaches one to recognize with considerable certainty the presence or absence of secundines. It is to be used in conjunction with the curette, but in my judgment is a far more reliable instrument for doing a complete job than is the curette which readily slides over membranes and even considerable masses of placenta without loosening them. In using the curette, one can safely make more pressure on the instrument if the fingers of the other hand are used to make counterpressure over the area being scraped. If the pregnancy is well advanced toward the fourth month, the cervix is easily dilated enough to admit the index finger, which is the most intelligent curette of all. With half the hand in the vagina the index finger is introduced into the uterus and the outside hand from above pushes the fundus down upon the finger so that every part of the cavity may be explored and the fragments of placenta loosened from their base. When the cavity is fairly clean the organ contracts and reduces the bleeding. The operation may be completed by washing out the uterus through a two-way catheter, with hot sterile water or various solutions, or the cavity may be swabbed out with a bit of gauze saturated with iodine. Rarely need any gauze be left in. If the organ does not contract and bleeding is too free it may mean that considerable placenta remains attached, and if circumstances admit, further efforts for its removal should be made. If while bleeding is too free it is thought best to terminate the procedure, then the cavity should be packed with iodoform gauze and ergot given.

After the fourth month of pregnancy, the above detailed method becomes less applicable, on account of the free bleeding which attends the procedure, and some method of starting labor pains is preferred. Such method will be the introduction of some foreign body into the lower uterine segment. If the cervix is long and uneffaced, metallic dilators may be gently used to prepare the way for the introduction of a rubber bougie or catheter and for the packing of the canal with gauze. I like to put the patient in the knee-chest posture (the Sims posture does very well), expose the cervix and vault of the vagina by the use of a Sims speculum to draw the perineum well back. Then seize the anterior and posterior lips of the cervix with a sponge or ring forceps. This gives an excellent view and control of the parts with which we have to deal. Iodine on a swab is applied to the cervix and canal and the adjacent parts protected by sterile or wet bichloride gauze. Now it is generally an easy matter to introduce a catheter or bougie freely anointed with sterile glycerine, into the cervical canal, by using forceps, without ever touching any other part of the operator or patient. I think this technic is to be much preferred over that of having the patient on her back, introducing two fingers into the vagina, and passing the bougie along the fingers and the vaginal wall into the cervix. Organisms which may give rise to infection are always to be found in the vagina, and even in the cervical canal, hence the less contact with these parts the better. Having introduced one or two bougies their full length, barring an inch or two, a strip of gauze may be pressed alongside of them into the cervix, and more loosely packed into the vagina to prevent the instruments from being forced out too soon. Following this the patient usually remains in bed, but there is no reason why she may not sit up if she can do so without discomfort. Labor pains sometimes set in promptly and may be promoted by the hypodermic use of pituitrin, which, however, will not start them. Generally the pains begin within twenty-four hours and continue in a natural way until delivery is accomplished.

Rarely this procedure fails to start a pain. Then further efforts must be made to bring about a delivery, for, having put one's hand to the plough, one cannot turn back. The next step is the introduction of a dilating bag of the pear-shaped type and appropriate size, to which a weight is attached. When this is expelled, if labor is not actively proceeding, some artificial method of delivery is in order, either the application of instruments or version and extraction. If the child is viable and not too near term, that is during the seventh and eighth months, vaginal Cesarean section affords a quick, if not easy way of accomplishing delivery. I would scarcely elect this method in a primipara, but in a multipara with a well dilated birth canal it is not a difficult operation, although a bloody one.

The greatest risk in all these procedures is that of infection; and in a general way it may be stated that the longer drawn out the undertaking, the greater the risk, or to speak more concretely, a prolonged tubing or bagging case is more apt to be followed by fever than is the more speedy curettement case. The latter I undertake without much concern, but the former I approach with profound respect.

REPAIRS FOLLOWING LABOR[1]

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INJURIES to the birth canal during the process of parturition will occur under the management of the most skilful as well as the most careless doctor; but there will be a marked difference in the number and the degree of the lacerations occurring in the practice of these two classes.

What measures can we adopt to minimize these injuries? The chief one I believe is to avoid undue haste. Do not resort too quickly to the use of instruments. I am sometimes called to apply instruments to a patient who has seemed to be in active labor a sufficient length of time to be delivered and on examination I find the cervix only partially dilated. If the mother's condition is good and the baby's heart sounds about normal in frequency, this is clearly not the time to use the forceps. Sedatives are indicated here. I confess, however, to have yielded more than once and, against my better judgment, have proceeded to dilate as well as I could and apply the forceps, because the patient and her anxious family have been prepared by the family physician to believe that as soon as the consultant arrived he would put the patient to sleep and terminate the labor.

Nature's method is to dilate and retract the cervix and we should wait until this process is well advanced before applying the instruments, unless there is a more urgent indication than the importunities of emotional relatives and a patient whose morale is at a low ebb. I believe that opium and scopolamine or belladonna are not used often enough in this condition. Injuries to the upper vaginal tract are not so amenable to treatment as the injuries lower down and hence are more likely to be followed by invalidism.

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[1] Read before the Kensington and Northeast Branches of the Philadelphia County Medical Society, October 23, 1919.

Laceration of the outlet can sometimes be prevented by delaying the birth of the head for three or four pains after it would seem that it must come through. This is accomplished by actually holding the head back and by directing the patient to breathe rapidly, or, as I tell them, to pant, when they have an almost imperative impulse to bear down. Rapid labor sometimes and instrumental labor often are the cause of injuries to the birth canal, and the question is, when and how shall we treat them?

It is my almost universal practice to deal with them at once. But if the condition arises in the middle of the night and sufficient light is not available to do accurate work, or if one does not have proper assistance, it is better to postpone the operation until the following day when competent help and light may be had. If the operation is delayed for five to seven days, the patient's getting up is postponed just so long, time which is practically lost by the patient. Only those patients in whom a deep cervical tear occurs which cannot be properly repaired at once, because of the thinning out of the cervix, gain by delay in operation and these form a small proportion of the whole number. Moreover, tears in the cervix are prone to heal without suturing if the wound is not infected, hence the cases are relatively few which would gain by delay in operation, if the operator is careful and skilful in bringing together the parts that belong together. It requires, however, more skill to coapt the tissues when they are relaxed, distorted, and perhaps edematous than it does when involution has advanced apace and the normal relation of the parts is more clearly defined.

It is my practice to inspect the birth canal for injuries as soon as the cord is tied and cut and the baby passed on to the nurse, not waiting for the delivery of the placenta. To do this I insert into the vagina the larger end of a large Sims speculum, pressing the cord behind it, and packing a bit of gauze in front and above to dam back the blood. This gives a clear view of the perineum and posterior vaginal wall without distorting them. I prefer this to the special dilators designed for exposing the torn parts, which not only expose but distort them. If the tear is not a serious one, I proceed at once to place the sutures and usually tie them before the placenta is delivered. Properly placed sutures will not tear out by delivery of the placenta.

I like to suture even small tears. Sometimes there is no lesion of the skin perineum but separation of the labia will disclose a quadrangular appearing tear at the base of the hymen, which one or two catgut stitches will suffice to close. If to this is added a central tear of the perineum not involving the sphincter ani, I prefer to use silkworm-gut sutures for tying on the skin surface, believing them to be less prone to infection, as they surely cause less reaction in the tissues than does catgut. If the laceration extends through the sphincter ani, I always make an immediate repair if the environment is suitable, that is, if material for aseptic work and competent assistance and light are at hand. I use a chromicized No. 1 buried suture in the sphincter muscle and silkworm-gut sutures, one or two of which include the sphincter ends, on the skin surface of the perineum. When the lesion extends up into the bowel I whip this over with a catgut suture which does not penetrate the mucous membrane of the gut but comes just short of it. If the primary repair of the wound is followed by cure—and this is usually the case—the patient is the gainer, whereas, if the wound breaks down through infection, the patient is no worse off than if no attempt had been made to cure her.

Lesions involving the sphincter are central, and of course, at the lower end of the birth canal, and usually do not extend high up. If, however, the injury has resulted from the use of high forceps, especially on occipitoposterior positions of the head, or other forced methods, the lesion may involve almost the whole length of the vaginal canal, including the cervix and the base of the broad ligament. Whether or not it is best to undertake the immediate repair of such an injury is debatable. Considerable hemorrhage is likely to result from it, and suturing is the most satisfactory way of dealing with this complication if operating room conveniences are at hand. But if the complication occurs in a private house, where competent help and material are not to be had, it is better to pack with antiseptic gauze and do an intermediate or secondary repair.

Not rarely the vulva is torn in some other place than the posterior commissure, it may be along the lateral walls, but is particularly prone to occur alongside of the urethra. These mucous membrane tears sometimes expose considerable raw surface and bleed freely, and I like to close them up with a fine running catgut suture. For the inside sutures I use catgut and for the outside ones, silkworm gut.

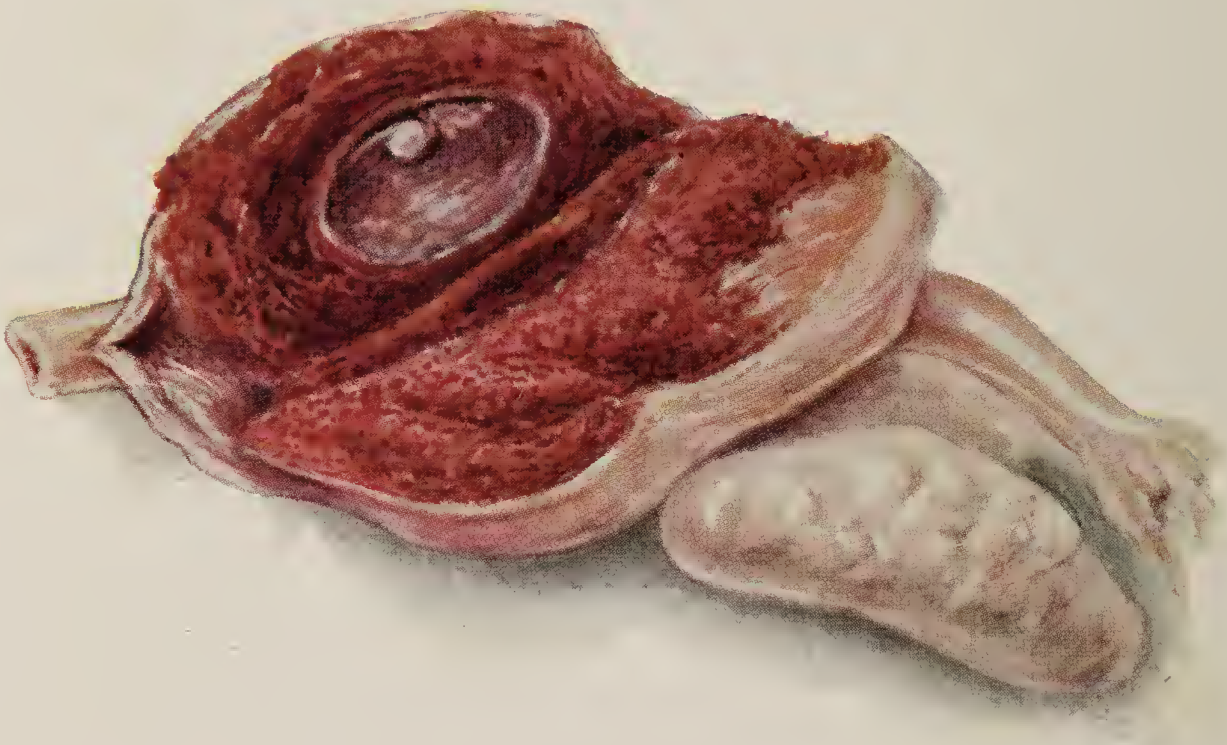

Plate III.—Extra-uterine pregnancy, embryo 10 mm. in length, approximate age 30 days.

VERY EARLY EXTRA-UTERINE PREGNANCY (30 DAYS)[1]

By ASTLEY P. C. ASHHURST, M.D.

SURGEON TO THE HOSPITAL

Margaret G., aged twenty-six years, was admitted to the Episcopal Hospital during the night of August 21, 1919, complaining of pain in the lower right quadrant of the abdomen. Her last menses, which began August 5, were normal. Eight days before admission (that is, August 13) she began to bleed again and to suffer some abdominal pain. On admission she was thought by the receiving ward intern to have salpingitis, and she was sent to the ward overnight. In the morning she appeared slightly anemic and a diagnosis of ruptured extra-uterine pregnancy (right) was made. The pain persisted and there was a very tender mass in the region of the right tube.

On opening the abdomen the presence of free fresh blood confirmed the diagnosis; the right tube was distended and tense with blood and bleeding from the fimbriated extremity continued. The tube and ovary (and, incidentally, the appendix) were removed, the blood evacuated and the abdomen closed. Recovery was uneventful.

Section of the tube discloses an embryo, lying lengthwise in the tube, in the midst of a blood-clot. (Plate III). The membranes were intact. Evidently tubal abortion was impending at the time of operation.

Dr. W. H. F. Addison, professor of histology and embryology in the University of Pennsylvania, very kindly examined the specimen under the microscope, and reported its length as 10 mm. The cephalic extremity was somewhat crushed, but the limb buds could be detected; they showed no indication of any digitations, nor even club-shaped expansion of their ends. From these data he estimated its age at about thirty days.

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[1] Specimen presented at a meeting of the Philadelphia Academy of Surgery, January 5, 1920. Reprinted from Annals of Surgery, 1920, i, 512.

1. PANHYSTERECTOMY FOR CHORIO-EPITHELIOMA; NO RECURRENCE SIX YEARS AFTER OPERATION

2. PANHYSTERECTOMY FOR FIBROIDS; CARCINOMA OF THE CERVIX DISCOVERED IN THE LABORATORY; NO RECURRENCE THREE YEARS AFTER OPERATION[1]

By ASTLEY P. C. ASHHURST, M.D.
SURGEON TO THE HOSPITAL

Dr. Ashhurst said that the two following cases were presented especially to emphasize the value of certain *measures of routine:*

1. Pathologic examination of uterine scrapings in cases of abortion or miscarriage.

2. Removal of the cervix along with the uterus in abdominal hysterectomy. In 3 out of the last 17 hysterectomies he had done for fibroids there had been coincident carcinoma of the cervix.

Chorio-epithelioma, or deciduoma malignum (Sänger, 1888), is an exceedingly malignant tumor growing in the body of the uterus after pregnancy. The pregnancy frequently is terminated before term, and the most favorable cases are those in which the diagnosis is made by the pathologist from examination of retained tissues removed in such cases. Such examination never should be neglected. The tumor probably arises from the chorionic and not from the decidual tissues; it behaves like the most malignant types of sarcoma, giving early venous metastasis, especially to the lungs (78 per cent.) and vagina (54 per cent.), according to Dorland.

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[1] Presented at a meeting of the Philadelphia Academy of Surgery, October 6, 1919. Reprinted from Annals of Surgery, 1920, i, 97.

1. PANHYSTERECTOMY FOR CHORIO-EPITHELIOMA; NO RECURRENCE SIX YEARS AFTER OPERATION

Agnes H., aged forty-two years, was admitted to the Protestant Episcopal Hospital August 9, 1913, for a recent abortion after a few weeks' pregnancy. The uterus was evacuated and the scrapings sent to the laboratory for examination, as a matter of routine. The pathologic report (Dr. C. Y. White) showing a chorio-epithelioma, abdominal panhysterectomy was done August 19, 1913. The appendix vermiformis, and left tube and ovary, the latter being cystic, were removed with the uterus, but the right tube and ovary were left. The uterus was slightly enlarged, and when opened, a papillomatous tumor was found at the fundus. (The specimen is illustrated in Fig. 1128 of the speaker's *Text-book of Surgery.*)

The patient has been kept under observation since operation, and is still in excellent health. Examination in August, 1919, six years after operation, disclosed no evidence of recurrence or metastasis. She is forty-eight years of age, and for the last year has complained of symptoms of the menopause.

2. PANHYSTERECTOMY FOR FIBROIDS; CARCINOMA OF THE CERVIX DISCOVERED IN THE LABORATORY. NO RECURRENCE THREE YEARS AFTER OPERATION

Cora L., aged thirty-six years, negress, was admitted to the Protestant Episcopal Hospital September 18, 1916, for profuse metrorrhagia. This was checked in the receiving ward by twisting on its pedicle a polyp which was protruding from the cervix. The patient's hemoglobin was only 25 per cent. On September 22, because slight fever persisted, she was etherized, the soft and dilated cervix was caught in volsellum forceps, drawn down to the vulva and the pedicle of the polyp, about 0.75 cm. in diameter and 6 cm. long, attached just above the cervix, was divided with scissors. The polyp itself was about 6 cm. in diameter. Very moderate bleeding occurred from the attachment of the polyp. The uterus was the seat of numerous fibroids.

October 6, 1916, two weeks after removal of the polyp, the patient's fever having subsided, and her hemoglobin having risen to 35 per cent., the abdomen was opened and complete panhysterectomy was done (*i. e.*, the uterus, including the cervix and both tubes and ovaries, was removed). The uterus was of medium size, containing several subperitoneal fibroids the size of hen's eggs or larger. Recovery was uneventful.

The laboratory report (Dr. C. Y. White) was that the cervix was the seat of *advanced epithelioma.*

The patient has been kept under observation, and now, three years after operation, pelvic examination discloses no symptoms of recurrence, nor is there any evidence of metastasis.

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